A MANUAL

OF

INFANTILE PARALYSIS

WITH

MODERN METHODS OF TREATMENT

INCLUDING

REPORTS BASED ON THE TREATMENT OF
THREE THOUSAND CASES

BY

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EPIDEMIOLOGIST, WISCONSIN, 1908, EPIDEMIC ACUTE POLIOMYELITIS

Copiously Illustrated With More Than One Hundred Engravings
Nearly All Original

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PREFACE.

In this volume we record our personal experience with acute poliomyelitis, its epidemic appearance, symptomatology, diagnosis, and methods of treatment. The manual is as free from technical terms as is possible in the discussion of a disease whose lesions involve the nervous system and the intricate functions of the body which the nervous system initiates, co-ordinates, and directs.

The most recent publication on the subject from the Rockefeller Institute of Medical Research is a monograph said to be based on the study of 161 cases of acute poliomyelitis. This number of cases of poliomyelitis is little more than the average number of cases seen daily in the Infantile Paralysis Department of the Hospital for Deformities and Joint Diseases. We regard the daily attendance for treatment of 100 to 150 children the best testimonial from the parents of the benefit derived by their own children, and the children of others who fall under observation as they await treatment.

The chapters on physical therapy and manipulative treatment are written in plain language, that the parents of a case may read what is needed for a child, and why. The spontaneous improvement which occurs in a certain percentage of cases may be augmented, deformities prevented, and cases with serious disability remedied, by the reasonable methods of treatment here outlined.

We have omitted reference to the obsolete and frequently misleading literature of the period which antedated the present pandemic of poliomyelitis. We have included numerous clinical descriptions, case reports, and autopsies,

consisting largely of cases of the more obscure and hitherto unrecognized variations of the eight classic forms of the disease, which have appeared in recent literature, and desire here to express our obligation to the several writers on whose material we have drawn to amplify and enhance the value of the manual.

We wish also to express our thanks to the publishers and editors of the medical journals in which a part of our work has already appeared for permission to reproduce some portions of such articles here.

We desire especially to thank those physicians to whom we are indebted for personal reports of cases.

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CHAPTER I.

Epidemics and Pandemics of Infantile Paralysis.

THE WISCONSIN EPIDEMIC OF INFANTILE PARALYSIS IN 1908.[1]

THE epidemic of spinal paralysis in Wisconsin during the summer of 1908 began in the vicinity of Eau Claire and spread out from there, as Dr. Chas. Harper, Secretary of the State Board of Health, told us, like circles in water.

When it became apparent the malady was extending through the State, the editor of the State Medical Journal requested reports of all cases sent in (to Dr. Manning) and inserted a blank in each copy of the journal for such report. Reports of 408 cases were received, and the undiagnosed and unreported cases would probably bring the number of cases in Wisconsin close to the 1000 mark.

The map of Wisconsin shows the involved areas. One hundred and sixty-seven of the cases occurred in Eau Claire City and County. In the northwest corner is a small group of 35 cases which occurred during the summer at Moose Lake, Minn. From these 408 cases I have selected 150 for clinical classification. Here are the brief histories of 2 cases illustrating the mild and serious forms of the disease:—

[1]An excerpt from the report read at the Sixty-third Annual Meeting of the Wisconsin State Medical Society, July 1, 1909, by Dr. Manning, Secretary of the Eau Claire County Medical Society. Reprinted from the Wisconsin Medical Journal of April and November, 1909.

Walter V. H., 12 years; July 30, 1908; predisposing cause, overexertion on a hot day, Buffalo Bill's circus in town; pain, vomiting, epistaxis, fever, headache; temperature 100° to 104° F.; no rash; paralysis of left leg only; reflexes lost; slight atrophy; no contractions; is now, Nov. 10, 1908, fat, strong, and well except motor paralysis of left leg. (Dr. D. B. Collins, Madison.)

Essie M., 10 years, female, no predisposing cause; no other cases in house, but two in neighborhood; pain in neck and back; severe headache; vomiting; temperature 99° to 103° F.; Sept. 16, paralysis of both legs, ascending involved arms, then trunk; reflexes absent; death third day; prostrated from time of onset; conscious; spinal type; no brain symptoms. (Dr. A. E. Bowles, Eleva.)

CLASSIFICATION OF 150 CASES OF POLIOMYELITIS.

Month of Occurrence.—January, 1; February, 0; March, 0; April, 0; May, 3; June, 4; July, 19; August, 44; September, 55; October, 21; November, 2; December, 1.

Age.—Less than one year, 10; one to five years, 64; six to fifteen years, 62; over sixteen years, 14.

Sex.—Males, 91; females, 59.

Other Cases in House.—This inquiry brought out 29 cases of direct exposure to infection; 25 of the children became ill during the time or immediately following a case in the same house. One boy (R. D.) slept in a tent with a boy who died of it four days later, the very day the boy (R. D.) was taken ill. One case had used milk supplied from a house where there was a case. Dr. Quade, of Wausau, reported a case occurring in a house in which a case had developed three years before; Dr. Cassidy, of Durand, reported a case which developed in a house in which a case occurred twenty years before. Seven cases occurring in the families of physicians have been reported.

Symptoms of Onset.—Fever. In the 95 cases in which the temperature is given, 84 had a temperature of 100°

Fig. 1.—Map of Wisconsin.

to 104° F.; 3, to 105°; 5 are said to have been "very high"; 3, from 98° to 99°.

Pulse. The pulse is given only twelve times, as it unfortunately was not called for in the blank; when given it is high, ranging from 100 to 168, and in fatal cases could be counted only by the stethoscope; in one case that is making a fair recovery, a child 7 years of age, the pulse remained at 160 for forty-eight hours after the onset, dropping to 120 when the paralysis appeared, when it gradually dropped to 78.

Nervous symptoms. Headache and backache, 66 times; pain and tenderness in limbs, 22; convulsions and convulsive movements, 26; tremor and inco-ordination, 2; cervical and spinal rigidity, 25; opisthotonus, 9; "stiff as a log from head to heels," 1; delirium, 12. Hyperesthesia of skin is often mentioned.

Digestive System.—Vomiting, 62 times, twice projectile in character; gastrointestinal irritation, 24 times.

Urinary Organs.—Retention, 11 times; suppression of urine, once; dysuria, twice; paralysis of sphincters, many times.

Skin.—A rash is noted 16 times; the writer has seen a rash in every case but one seen in the acute stage. Herpes labialis, once.

Distribution of Paralysis.—One lower extremity, 26; both lower extremities, 46; two lower and one upper, 4; both lower, one arm, and dyspnea, 2; both lower and sphincters, 3; one arm and one leg, 3; all extremities, 6; facial only, 2; general paralysis, 12; deltoid and shoulder, 2; sternomastoid, 6; left peroneus and bladder, 1; comatose for several days, no paralysis, 2; paresis only, 6; paralyzed, no further data, 11; fatal cases, 22.

The map of Eau Claire shows the neighborhoods which in succession were invaded.

In the city of Eau Claire, in a small area of the ninth ward, there were 9 cases and 3 deaths the last week of July. Following these cases a number promptly appeared scattered pretty well over the town, but each early case in a neighborhood was followed by several in that immediate locality.

On looking back through the death certificates on file in the office of the Board of Health, I found the death reported, on May 29th of that year, of a girl aged 7 who lived just across the river from these cases, and at the east approach of the same bridge. The death certificate read: Cause of death, "measles; contributing cause, epilepsy." She had been sick three days. The family said she had never had a fit before. The Norwegian physician, who was called to the case a very few hours before death occurred, admitted to me that it was probably an undiagnosed case of poliomyelitis of the fulminating type.

Further investigation of the death certificates showed that a young man of 20 years had died of convulsions in this same neighborhood on July 16th. He was sick less than one day, and no doctor had been called to the case till death occurred.

The last distinct group of cases to appear was in the third ward. This is the aristocratic residence portion, and is somewhat separated from the rest of the town.

The first case in this area appeared September 5th, and heralded 15 cases and 5 deaths. Although, as I said, this is the aristocratic section of the town, a wagon road crosses it from the farming districts to the east of the city. On this wagon road a watering trough for stock was placed and a man of 50 years of age opened a small shop nearby, where all of the children ran for bonbons. The first 3 cases were one-half block from this shop, three

Fig. 2.—Distribution of infantile paralysis in 1908, in the city of Eau Claire, Wis., taken from *Dr. Jacolyn Manning's* Report to the Wisconsin State Medical Society at Madison, Wis., July 1, 1909.

boys in three different homes, each of which developed a paralysis, but recovered. One block south on the same street, 2 brothers developed the disease on Wednesday and Thursday preceding the Sabbath on which they both died within five hours. Called in consultation I witnessed these 2 deaths, the termination in each case due to an ascending paralysis. Fourteen of the 15 cases were chil-

Fig. 3.—Map (diagrammatic) of United States.

dren and young adults, but the fifteenth was the man of 50 who kept the little shop. On October 21st he was taken ill, and said to a neighbor who found him sitting over a stove, "I have had backaches before, but never anything like this." He developed a paralysis of both legs and died October 26th.

Twenty miles south of Eau Claire is the village of Augusta. Three miles from Augusta live the Wagner family, consisting of father and mother and 6 children. Their farm is a clearing in the woods. They grew corn

and potatoes, lived in a one-room log house, and Wagner worked steadily clearing his little domain, exchanging firewood for groceries and clothing. They had no pump, carrying water from the creek one-fourth of a mile away. In the summer of 1908 they took a boarder, an old man who was a county charge, for whom they received the sum of $1.50 a week. A few days after the old man's

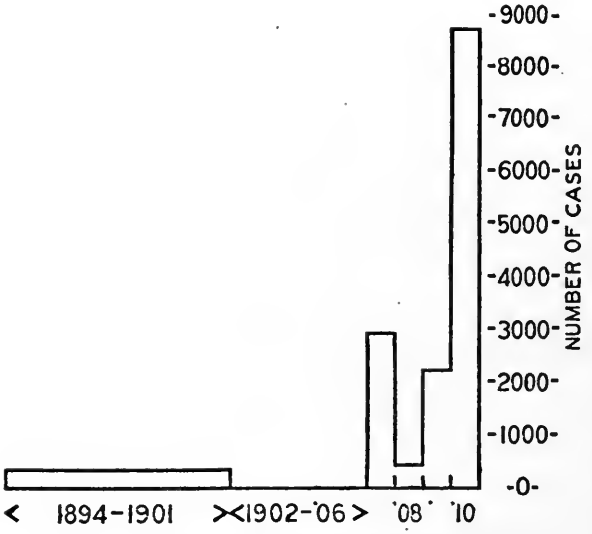


Fig. 4.—Prevalence of cases in United States.

arrival he was taken ill. Mrs. Wagner nursed him, but a week later her husband became ill in the same manner; and one by one all of the children. The old man and 3 of the children got well. The father was left with a paralysis of one leg and 3 of the children had an arm or leg paralyzed—the girl aged 10, a boy 4 years of age, and a baby.

In the report a review of the epidemics of acute poliomyelitis in Norway and Sweden, with the occurrence of many hundred cases during the four summers of 1903-4-5-6, is given, with a review of the epidemic in and about

the port of New York, "our principal port of entry from Europe," and the article concludes: "It would seem that a highly contagious disease had journeyed along the main highways of travel from the Old World to the Eastern United States, and thence to these sections of the Middle West, where there is a large percentage of Scandinavians residing."

"There were about 8700 cases of infantile paralysis reported in the United States in 1910, and if we contrast that figure with the years prior to 1904, when the average yearly number was 15 cases per year, we may appreciate what an enormous increase has occurred.

"When we come to the analysis of the figures for 1910 we must remember that they were brought out by a systematic and extended inquiry undertaken by the Massachusetts State Board of Health, and probably had such an inquiry been possible in one of the earlier years a larger number of cases would have been discovered than now stand as reported in those years. Still, one cannot conceal the fact that in 1910 there apparently occurred a very much larger number of cases than before all over the United States, and that the extent and distribution of the disease in this country in 1910 was of a different character from that of any previous year." (Lovett.)

COINCIDENTAL EPIDEMIC PARALYSIS IN ANIMAL AND MAN.

Epidemiologists of the recent epidemics of poliomyelitis in man have not overlooked the coincidental paralysis and death among domestic animals, yet the aggregate of such occurrences has not hitherto, I believe, been assembled.

A close relationship between paralytic cases in man and animal during epidemics of poliomyelitis has been

observed in the United States, and in Sweden, Westphalia, England, and Brazil.

ACUTE PARALYTIC DISEASE AND DEATH AMONG DOMESTIC ANIMALS OCCURRING COINCIDENTLY WITH EPIDEMIC POLIOMYELITIS IN MAN. (MANNING.)

Reported by	Locality	Year	Horse	Sheep	Dog	Cat	Hog	Fowls	Total
Caverly	Vermont	1894	Horse		Dogs			Chickens	Many
Wickmann	Sweden	1905			Dogs				And other animals
Free	Dubois, Pa.	1907					Pigs	Chickens	
Manning	Wisconsin	1907-08	Colts	Sheep		Cats		Ducks	Many
Lovett	Massachusetts	1911	Horse		Dog	Cats		Hens	39 in all
Hill	Minnesota	1909	Colts						Three
Snow	California	1910	Colts		Dog	Cats		Chickens	Many
Kelley	Washington	1910			Dog paralyzed 1 week before onset in child				
Williams	Wash'n, D. C.	1910						Chickens	Many
Bierring	Iowa	1910				Cat	Hog	Chickens	Many
King	Indiana	1911	18 animals (1 cow) paralyzed among 102 cases of poliomyelitis						
Batte	Ohio-Ky.	1911						Chickens	
Krause	Westphalia	1910						Chickens	Many
Gregor & Hopper	Cornwall, England	1911	Horse, one week before onset of paralysis in boy						
Carina	Sao Paulo, Brazil	1910-11	1000 horses and 4000 cattle dead with "symptoms of rabies," coincidental with 13 cases of human poliomyelitis at Sao Paulo						
N.Y.Tribune Oct. 1, 1912	Kansas & Nebraska	1912	Horse; meningitis, paralysis, death, 24,000. Hostlers said to contract disease						
Langhorst	Illinois	1912	Dog						

In the State of Massachusetts in 1910, during an epidemic of 1000 cases of poliomyelitis, paralysis among horses and cattle included many colts, geldings and horses, and also many heifers, cows and bulls. One veterinarian attended 15 cases of paraplegia in cows (Wentzell, Beverly) of which most died. Three other paralytic cows are said to have had twisting of the neck, circled to the right; when forced to walk, complete paralysis of hind quarters. A Guernsey bull with complete paraplegia was killed six weeks later, when atrophy of gluteal muscles was noted. (May.)

The following cases of sickness, paralysis or death among small animals and fowls was noted during the same season:—

Paralysis in Domestic Animals.—The table shows that out of 186 families in which acute epidemic poliomyelitis occurred, 34 homes had illness, paralysis or death in 82 animals. One hundred and ten

of the families above mentioned had animals; therefore, about 30 per cent. of 110 families with animals had illness, paralysis or death in their animals. (Springfield, Mass., 1910. Sheppard.)

DATA AS TO DOMESTIC ANIMALS.

	Families.
No animals of any kind in	76
Animals present in	110
	186

6 homes had 14 hens with sickness.
6 homes had 6 cats with sickness.
4 homes had 4 dogs with paralysis.
4 homes had 12 hens with paralysis.
2 homes had deaths in 2 dogs.
6 homes had deaths in 6 cats.
8 homes had deaths in 42 hens.
2 homes had deaths in 2 horses.
34 homes had illness, paralysis or death in 82 animals.

At Penryn, Cornwall, England, on May 27, 1911, a boy of 6 years of age was taken acutely ill; he was observed falling about the house; he went out and again fell and was carried home, and paralysis of all extremities followed. This was the second of 132 cases of paralysis in Cornwall and Devon, the adjoining shire, during the same summer. One week before the boy's illness a horse belonging to this lad's father had an attack of what is locally known as "poke-neck;" it is said to have been paralyzed in the neck and forequarters; it fell down in the stable and was unable to rise; when taken out of the stable with assistance it again fell; it was shot without having been seen by the veterinary surgeon.

June 8th, a lad of 2½ years became ill with a paralysis of both legs. The second boy's father had been in the house with the first case. (Gregor & Hopper, "Poliomyelitis in Cornwall," British Med. Jour., Nov. 4, 1911.)

Reading of this report of coincidental paralysis in man and horse, occurring in the same homestead during a considerable epidemic of poliomyelitis, crystallized a belief that has been constantly augmented during four years'

study of this disease. The season preceding the Wisconsin epidemic of 1908 I was asked to examine two recently foaled colts on the stock farm of Chas. L. Allen, of Eau Claire, Wis. These colts were affected with a spastic paralysis, all four legs sticking rigidly forward; when lifted to a foothold they could not stand. They were two of a considerable number of blooded colts that were lost that season. A young riding horse also developed a spastic gait and had to be disposed of.

During the epidemic of 1000 cases in Minnesota in 1909 three colts were seen by Dr. H. W. Hill, epidemiologist of the Minnesota State Board of Health, ill with a disease "strongly analogous in clinical history and symptoms to the disease in the human." (Hill, Minn. Med. Jour., Sept. 1, 1909.)

These colts were under the care of Dr. C. S. Shore, a veterinary surgeon of Lake City, Minn., who wrote of them the following excellent clinical record:—

In my veterinary practice of the past five or six years I have found a disease appearing among one- or two- year-old colts that shows a line of symptoms corresponding closely to anterior poliomyelitis in children. I have had from 5 to 10 cases a year during this time, always occurring during the summer months, and the majority of them during the month of August. The affected colts are usually found in the pasture unable to stand. The owner will sometimes notice an unsteady gait for twenty-four hours before entire loss of motion occurs. At first the colts have a rise of temperature to 104°; pulse and respiration accelerated; animal sweats profusely; there is some trouble noticed in swallowing, especially water; bowels tending toward constipation; more or less tympanites; retention of urine for a few hours at least. Head drawn back so the end of the nose tends to assume a position somewhat on the line with the neck.

The death loss is less than 10 per cent., but in those that recover the market value is depreciated, because of faulty gait the animal assumes after an attack of the disease; there is atrophy and contraction of certain muscles or certain groups of muscles. It seems that

the flexor muscles of the legs especially are more often affected than the extensor, and in almost all of the cases some of these deformities are likely to remain permanent, causing a flexion of the fetlock. The elevators of the head are also likely to become affected, causing the head to have a poky appearance as it is carried out from the body.

After one of these attacks the colt will remain down from one to three weeks, and will then continue to improve for a year, but it seldom if ever makes a complete recovery. (Bulletin Mass. Board of Health.)

Dr. Shore's interesting note that the colts had difficulty in swallowing will be referred to again later. It is evident these colts were affected by the same disorder as the horse at Penryn, which died after developing poke-neck and paralysis of the forequarters.

The State of California had an epidemic of 100 known cases of poliomyelitis in 1910. The majority of these cases occurred in San Joaquin County, and according to the September, 1910, Bulletin of the California State Board of Health, "veterinarians report a considerable number of puzzling paralyses of colts in San Joaquin County, where the largest number of cases have occurred so far."

Historical Cattle Plagues.—At Echternach in the Luxembourg there is an annual dance through the church, of pilgrims, headed by the clergy, to the shrine of St. Willibrod. The pilgrimage is done by way of vows for the cure of nervous diseases. The local legend asserts that the ceremony had its origin in a cattle plague which began in the eighth century, which ceased through an invocation to the saint. The dance is headed by the clergy and proceeds to a traditional tune from the banks of the Sûre to the church, up sixty-two steps, along the north aisle, around the altar with the sun, and down the south aisle. It is curious that until the seventeenth century only men took part in it. ("Medieval Stage," E. K. Chambers, 1911, vol. i, page 163.)

A knowledge of this plague, simultaneously affecting animals and man with an acute nervous disease, reappearing at irregular intervals through the ages, and ceasing (?) on an invocation to the saints after all the susceptibles had been killed or crippled and an immunity established for the rest of the inhabitants, sheds a flood of light on the so-called miracle cures of the middle ages and today. A thousand pairs of crutches might well adorn the walls of a church which could cure (?) paralysis of the legs, but it is to be feared the cure was inoperative where the paralysis was not regressive in type.

Pigs and Chickens.—The reports of paralyzed fowls from districts where poliomyelitis exists are common, and call to my mind a number of similarly affected hens I saw some years ago on the John Seymour ranch in Allen County, Kansas. The housewife reported the death of a hundred hens from an epidemic disease, and called my attention to one of those remaining which crouched on the ground, and when disturbed fluttered a short distance, dragging its feet. Other fowls were unable to use one wing; many chickens whirled wildly about until they fell dead. The epidemic was clearly an acute nervous disorder, producing ataxic and paralytic types.

Examination of Paralyzed Chickens.—(*a*) Dr. Charles L. Dana examined one of the fowls paralyzed during the Vermont epidemic of 1894, and found, "an acute poliomyelitis of the lumbar portion of the cord; no meningitis; bacteriological examination negative." (Caverly, *loc. cit.*)

(*b*) Dr. F. A. Ely examined a paralyzed chicken with a most suggestive history from a Boone County, Iowa, farm. The chicken had an acute illness not exceeding three days in duration, and one wing and both legs were paralyzed. During the summer many of the chickens on this farm became ill and disabled, and as soon as this was noticed the farmer would snap off their heads and throw the chickens into the hog-yard. Some time later a large hog developed a

typical paralysis of both hind legs, so that the animal wore the skin off his knees as he dragged himself about. In October, 1910, Dr. Ely saw in consultation a child with acute poliomyelitis on this farm; at the same time of Dr. Ely's visit, another case of paralysis was observed in a chicken which was taken to Des Moines for examination.

Dr. A. R. Robertson, pathologist at Drake University Medical School, reported: "Examination of fowl paralyzed after three days' acute illness (one wing, both legs). Upon exposure of the spinal cord, a distinct area of softened cord, one inch in length, of the lower dorsal and upper lumbar regions was observed. Histological sections from the affected areas revealed numerous small hemorrhages in the anterior cornua, and distinct collections of cells in perivascular and perilymph channels, and tissue spaces of the anterior horns. The histological picture was that of acute poliomyelitis in man." (Bierring, "Acute Poliomyelitis in Iowa in 1910-1911," Interstate Med. Jour., Jan., 1912.)

At Pella, Marion County, Iowa, one hundred and fifty miles distant from the Boone County case, a similar association between a paralytic disease in chickens and cases of acute poliomyelitis was noted. A number of these chickens were obtained. Three of them were kept for two, three, and four weeks, until the paralysis and a certain degree of muscular atrophy were established, and then examined, with confirmatory results. (Bierring, *loc. cit.*)

In September, 1911, 5 cases of acute poliomyelitis in Calhoun County, Iowa, 3 of which proved fatal in the first forty-eight hours, occurred on four different farms. On the farm of each of 4 of the cases a history of paralytic disease in lower animals was obtained. In 2 instances chickens were affected; kittens at one farm, pigs at another. (Bierring, *loc. cit.*)

The Dog.—The coincidental paralysis of the dog and human poliomyelitis has been reported from Sweden, Vermont, Massachusetts, Illinois, California, and the State of Washington. Kelly, of the State of Washington, reported that one family had 1 dog paralyzed for two days one week before onset in child (1910). Langhorst, of Illinois, reported 2 cases of acute poliomyelitis succeeding paralysis in a dog with which patient associated. This

report is given in full under "Transmission of Acute Poliomyelitis through the Dog" in Chapter II of this volume.

The State of Massachusetts had an epidemic of 923 cases of poliomyelitis in 1909 and a second epidemic of 845 cases in 1910. The summer of 1910 the following cases of paralysis in dogs were collected by the investigator employed by the State Board of Health:—

PARALYSIS IN MASSACHUSETTS DOGS, 1910. (MAY.)

Dedham. French bulldog, paraplegia gradually working forward, death.

Newtonville. French bulldog, paraplegia. Recovered in three days.

Amesbury. Twelve-year-old housedog. Complete paralysis, posterior to lumbar region, sudden and continuing.

Boston. Nine-year-old housedog, paraplegia.

Boston. Wadsworth. Three dogs of different breeds and ages; all had same symptoms: ascending paralysis of all four legs, coma and death. Head twisted to right. Two lived for twelve days. The puppy lived three days.

Boston. Cocker spaniel. October 11th, complete paraplegia; Dec. 5th, dog able to walk and improving.

Salem. Several cases of paraplegia in dogs, accompanied by vomiting; all fatal.

Reading. Six cocker spaniels from 4 to 7 years of age. Paraplegia, some very sensitive to touch. Recovery.

Boston. May. Spaniel. Slight paraplegia developing complete paralysis of hind legs, bowels and bladder in August, 1905. Paralysis and atrophy present in 1910.

Sheep.—"In England paralysis is epidemic among sheep in the early autumn each year." ("Torment of Flies," Shipley, Christ College, Cambridge.) It is well known that Sir Walter Scott was afflicted with a short and withered leg. He had an acute illness when he was a small lad which left him unable to walk, and he was given in charge of an old shepherd of whom he was very fond.

This shepherd took the boy out in his plaiddie to the fresh air of the sunny hillsides, and health returned to him, but he was left with a paralysis of one leg.

In the fine study of the life of the English shepherds of today, W. H. Hudson relates the story of an ataxic sheep, told by the shepherd, Caleb Bawcome, who was himself the victim of poliomyelitis. The description of man and sheep is as follows:—

"Caleb, a shepherd of the downs. A very tall, big-boned, round shouldered man, uncouth to grotesqueness, who walked painfully with the aid of a stick, dragging his shrunken and shortened bad leg. . . . . He told me that when he were a young man he was once putting the sheep in the fold, and there was one that was giddy, a young ewe. She was always turning round, and round, and round. And when she got to the gate she wouldn't go in, but kept a'turning and a'turning 'till at last he got angry and, lifting his crook, gave her a crack on the head." He tells of other giddy sheep, "giddy because they had a maggot on the brain, or some other trouble I couldn't find out." He also tells of an unlucky farmer in those parts whose sheep fell sick and died in numbers, year after year, bringing him down to the brink of ruin. ("A Shepherd's Life," W. H. Hudson, pages 52, 127, and 352.)

Here, then, is a record of an epidemic sheep plague occurring annually; a very good description of an individual case of the ataxic type occurring in a young ewe; the shepherd presenting the typical flaccid paralytic type; and our knowledge of Walter Scott, the child of aristocratic parents, contracting the acute disease coincidently with association with a shepherd.

A TABLE OF EPIDEMICS AND PANDEMICS OF ACUTE POLIOMYELITIS. COMPILED FROM MANY SOURCES BY DR. JACOLYN V. V. MANNING.

Year.	Locality.	Cases.	Deaths.	Reported by
1841.	Louisiana	11	....	Colmer, Am. Jour. Med. Sc., Jan., 1843.
1868.	Norway	14	....	Bull.
1875.	Philadelphia (in 4 years)	86	....	Sinkler, Bost. Med. Surg., Nov. 23, 1898.
1881.	Umea, Sweden	13	....	Bergenholz-Medin. Intrn. Med. Con.
1885.	S. Foy, Germany	13	4	Cordier, Lyon méd., 1888.
1886.	Mandel, Norway	9	....	Oxholm-Leegard, Neur. Centrb., 1890.
1887.	Stockholm, Sweden	43	3	Medin, Nord. Med. Ark., 1896.
1893.	Boston	26	....	Putnam, Boston Med. Jour., 1893.
1893.	St. Girons, France	9	....	Andre, Compt. de méd. Bordeaux.
1894.	N. Adams, Mass.	10	....	Brackett, Tr. Am. Orth. Asso., xi, 132.
1894.	Rutland, Vermont	132	18	Caverly, N. Y. Med. Record, 1894.
1895.	Spertoli, Italy	7	....	Pericinni, La Sperimental, 1895.
1895.	Genoa	6	....	Bucelli, Policlinico, 1895.
1895.	Stockholm, Sweden	20	....	Medin, Nord. Med. Ark., 1896.
1896.	Much Haden, England	7	1	Pasteur, Tr. Clin. Soc., 1897.
1896.	Port Lincoln, Australia	18	....	Altman, Austr. Med. Gaz., 1897.
1896.	Cherryfield, Maine	7	1	Taylor, Phil. Med. Jour., 1898.
1896.	Greene Co., Ala.	15	....	Bondurant, Phil. Med. News, 1901.
1897.	London	11	....	Buzzard, Lancet, 1898.
1897.	Kiel, Germany, Baltic Sea	4	....	Pleuss, Inaug. Diss., Kiel, 1898.
1897.	New York City	12	....	H. L. Taylor, N. Y. Med. Jour., 1897.
1898.	Le Grand, Cal.	4	....	Newmark, Med. News, Phil., 1899.
1898.	Royersford, Pa.	22	....	Jour. Nerv. and Ment. Dis., 1899.
1898.	Vienna	208	....	Zappert, Jahrb. f. Kinderheil., 111.
1898.	Frankfort on Main	9	....	Auerbach, Neur. Centralbl., 1900.
1899.	Stockholm, Sweden	54	3	Wickmann, Heine Med. Krank., 1907.
1899.	Bratsburg, Norway	54	2	Leegard, Norsk Mag. f. Laeg., 1901.
1899.	Poughkeepsie, N. Y.	37	1	H. D. Chapin, Arch. Ped., 1900.
1900.	Gloucestershire, Mass.	52	....	Painter, Trans. Am. Orth. Asso., 1902.
1901.	San Francisco, Cal.	55	....	Woods, Occidental Med. J., xvii, 77.
1903.	Gotteburg, Sweden	20	1	Wickmann, Heine Med. Krank., 1907.
1903.	Norway	18	6	Harbitz, Jour. Am. Med. Asso.
1903.	Parma, Italy	26	....	Lorenzelli, La Pediatria, 1904.
1905.	Queanbergen, N. S. W.	6	....	Blackhall, Austr. Med. Gaz., 1904.
1905.	Sydney, Australia	25	....	Litchfield.
1905.	Stanmore, Australia	34	....	Wade.
1905.	Brisbane, Queensland	108	4	Ham, Austr. Med. Gaz.
1904.	Hvalen, Norway	41	....	Nannsted, Norsk Mag. f. Laeg., 1906.
1904.	Aafjordan, Norway	20	6	Platon, Tidssk. f. d. Norsk, 1905.
1904.	Trondheim, Norway	437	67	Giersvold, Norsk Mag. f. Laeg., 1906.

A TABLE OF EPIDEMICS AND PANDEMICS (*Continued*).

Year.	Locality.	Cases.	Deaths.	Reported by
1904.	Sweden	1031	....	Wickmann, Tidssk. f. d. Norsk. 1906.
1905.	Norway	952	111	Harbitz, Jr. Am. Med. Asso., Sept. 7, 1912.
1906.	Norway	466	50	Harbitz, *ibid.*
1907.	Norway	204	30	Harbitz, *ibid.*
1905.	St. Louis, Mo.	....	....	Fry.
1905.	Central Illinois	8	....	Norbury.

1906. (During the year 1906, curiously free from reports of poliomyelitis in the city of New York, there was said to have been a serious epidemic of cerebrospinal meningitis. Contrary to the history of the spread of epidemics of all ages, the epidemic of 3000 cases of poliomyelitis in the year 1907 is supposed to have appeared like a bolt from the blue.)

1907. First American Pandemic.

Year.	Locality.	Cases.	Deaths.	Reported by
1907.	New York City	2500	....	New York Committee.
1907.	Schenectady	29	....	Clow, Alb. Med. Jour., 1908.
1907.	Oil City, Lehigh, Dubois and Ridgway, Pa.	209	....	Urey and Terriberry.
1907.	Massachusetts	234	....	Lovett, Mass. State Bul.
1907.	Live Oak, Florida	16	....	Efird, Tr. Flor. State Asso., 1908.
1907.	Oceana Co., Mich.	20		
1907.	Trempealeau Co., Wis.	22	....	H. A. Jegi, Tr. T. P. C. Co. Med. Soc.

1908. Second American Pandemic.

Year.	Locality.	Cases.	Deaths.	Reported by
1908.	Massachusetts	136	....	Lovett, Mass. State Bul.
1908.	Clearfield, Pa.	14	....	Mills.
1908.	Salem, Va.	25	....	Wiley and Darden.
1908.	Florida	16	....	Frost.
1908.	Flint, Mich.	30	....	Manwaring, of Flint, Mich.
1908.	Wisconsin State reported	408		
	(est.)	1000	....	Manning, J. V. V., Secretary Eau Claire Co. Med Society, Wis. Jour. Med., April and November, 1909.
1908.	Minnesota	150	....	A. S. Hamilton, Neur. Dept. Univ. of Minnesota.
1908.	Whitmore, Iowa	9	....	A. S. Hamilton, *ibid.*
1908.	Missouri	....	....	Frost, Public Health Bul., 44.
1908.	Victoria, Australia	155	7	Stephens, Inter. Co. Med. J., 1908.

A TABLE OF EPIDEMICS AND PANDEMICS (*Continued*).

1908 AND 1909. FIRST EUROPEAN PANDEMIC.

Year.	Locality.	Cases.	Deaths.	Reported by
1908.	Russia (a village of 500 inhabitants)	49	....	Schwarz, St. Petersburg med. Woch., Jan., 1909.
1908.	Germany	1000		
1909.	Westphalia	100	....	Krause, Deutsch. med. Woch., 1909.
1909.	Rhenish Westphalia	500	....	Rekseh, Med. Clin., 1909.
1908.	Essex, England	8	....	Treves, Brain, Nov., 1909.
1909.	Leyden, Holland	24	....	Netter.
1909.	Spain	9	....	Netter.
1909.	France	6	....	Netter.
1909.	Zurich, Switzerland	3	....	Eichhorst, Corres. Bltt., Basel.

1909. THIRD AMERICAN PANDEMIC.

Year.	Locality.	Cases.	Deaths.	Reported by
1909.	Massachusetts	923	....	Lovett, Mass. State Bul.
1909.	Brooklyn, N. Y	150	....	Le Grand Kerr.
1909.	New Jersey (est.)	200	....	Keppler.
1909.	Santa Clara, Cuba	140	....	Off. Sanitary Bul. of Havana.
1909.	Maryland	a few	2	Corresp. Sec. State B. of H.
1909.	Minnesota	1100	238	Corresp. Sec. State B. of H.
1909.	Nebraska	999	244	Corresp. Sec. State B. of H.
1909.	Kansas	100	....	Corresp. Sec. State B. of H.
1909.	California	16	....	Bulletin of the State B. of H.
1909.	Oregon	55	11	Corresp. Sec. State B. of H.
1909.	N. Dakota	75	25	Corresp. Sec. State B. of H.
1909.	Montana	....	4	Corresp. Sec. State B. of H.
1909.	Indiana	....	14	Corresp. Sec. State B. of H.
1909.	Richland Center, Wis.	18	....	Harper, Sec. Wis. State B. of H.
1909.	Illinois	....	....	Frost.

1910. FOURTH AMERICAN PANDEMIC OF POLIOMYELITIS.

(The United States Census Bureau returns for 1910 give 1459 deaths reported due to infantile paralysis; if the mortality is considered to average 10 per cent., the estimated number of cases in the United States in 1910 would reach the sum of 14,590. Lovett has compiled 8700 cases in the United States in 1910.)

Year.	Locality.	Cases.	Deaths.	Reported by
1910.	Arizona	30	....	Lovett, Inf. Par. in U. S. in 1910.
1910.	California	100	....	Lovett.
1910.	Colorado	....	4	Corresp. Sec. State B. of H.
1910.	Connecticut	168	....	Lovett.
1910.	Delaware	8	....	Lovett.
1910.	District of Columbia	500	....	Williams, T. A.
1910.	Florida	19	....	Lovett.
1910.	Idaho	96	....	Frost.
1910.	Illinois	137	....	Frost.
1910.	Indiana	500	....	Lovett.
1910.	Iowa	654	157	Bierring, W.
1910.	Kansas	198	....	Lovett.
1910.	Kentucky	3	....	Batte, Dr. John, Cincinnati.

A TABLE OF EPIDEMICS AND PANDEMICS (*Continued*).

Year.	Locality.	Cases.	Deaths.	Reported by
1910.	Louisiana	50	....	Lovett.
1910.	Maryland (est.)	300	....	Lovett.
1910.	Massachusetts	843	....	Lovett.
1910.	Michigan, Hillside	72	12	Green, Dr. B. F., of Hillside.
1910.	Minnesota	1000	....	Lovett.
1910.	Montana (est.)	170	....	Lovett.
1910.	Nebraska	144	....	Corresp. Sec. State B. of H.
1910.	Nevada	9	....	Lovett.
1910.	New Hampshire (est.)	210	....	Lovett.
1910.	New York reported	225		
	(est.)	460-500	....	Frost.
1910.	N. Dakota (est.)	150		
1910.	Ohio	50	....	Frost.
1910.	Oklahoma	20	....	Lovett.
1910.	Oregon	112	....	Corresp. Sec. State B. of H.
1910.	Pennsylvania	1006	....	Corresp. Sec. State B. of H.
1910.	S. Carolina	150	....	Bul. S. C. State B. of H.
1910.	Rhode Island	231	23	Frost.
1910.	S. Dakota	86	....	Lovett.
1910.	Utah (est.)	120	....	Lovett.
1910.	Vermont	48	....	Corresp. Sec. State B. of H.
1910.	Virginia	335	....	Corresp. Sec. State B. of H.
1910.	Washington	225	12	Corresp. Sec. State B. of H.
1910.	Wisconsin (est.)	370		
1910.	Montreal, Quebec	38	....	Colin Russel, Montreal.
1910.	Ontario, Canada	179	....	Lovett.
1910.	British Columbia	75	....	Lovett.
1910.	Schleswig-Holstein	132	....	Meyer.

1911. (The United States Public Health Report for April, 1912, stated the returns for 1911: Poliomyelitis, cases, 1930; deaths, 440.)

Year.	Locality.	Cases.	Deaths.	Reported by
1911.	New York City, endemic	....	....	Manning.
1911.	Indiana (to October 28)	102	17	King.
1911.	Virginia Mountains	25	....	Evan Evans.
1911.	Louisiana	20	....	N. O. Times Democrat.
1911.	Iowa	68	12	Bierring.
1911.	Missouri	....		
1911.	Arkansas	....		
1911.	Oklahoma	....		

1911. European Pandemic.

Year.	Locality.	Cases.	Deaths.	Reported by
1911.	Sweden	3840	....	Kling and Petterson.
1911.	Norway	1250	125	Harbitz, *ibid.*
1911.	Denmark	250	....	Thomsonn, Hospitalstidend, liv, 1329.
1911.	England	150	....	Gregor and Hopper, and others.
1911.	Switzerland, 3 Districts	....	....	Hagenbach, Basel.
1911.	Germany	1000	....	Slomann, Copenhagen.

A TABLE OF EPIDEMICS AND PANDEMICS (*Concluded*).

1912. UNITED STATES.

Year.	Locality.	Cases.	Deaths.	Reported by
1912.	Massachusetts (August) ...	....		
1912.	New York City, endemic (August)	....	....	Manning.
1912.	Buffalo (August)	....	300	
1912.	California (Los Angeles) (August)	....		
1912.	Norway	117	* 5	Harbitz, *ibid.*
1912.	Sweden (from Jan. to Aug. 15)	1458	....	Kling and Petterson.

CHAPTER II.

The Etiology or Exciting Cause of Infantile Paralysis.

FOUR hypotheses have been advanced to explain the nature of the hitherto unknown cause of infantile paralysis:—

I. A chemotoxin.
II. Geirsvold's bacterium, a micrococcus.
III. Flexner's filterable, ultramicroscopic virus.
IV. Dixon's protozoön.

I. *Chemotoxin.*—It was formerly held as a theory that acute poliomyelitis was induced by the action of a chemical toxin ingested in the form of decomposed animal matter, and was, in fact, a sort of ptomaine poisoning. This theory has never been supported by scientific investigation, and fell to the ground when it was demonstrated that the disease could be transmitted from one living host to another, with no diminution of its toxicity.

II. *Geirsvold's Bacterium or Micrococcus.*—In the fall of 1905 Geirsvold, of Norway, demonstrated the presence of the same micro-organism in a whole series of cases of acute poliomyelitis. The organism was obtained three times by direct examination and twelve times by cultural methods from the cerebrospinal fluid. A pure culture which readily produced chains of four or six elements was grown in nutrient broth. The organism which he described was a bean-shaped diplo- or tetra- coccus, with certain cultural characteristics. Geirsvold produced paralysis in mice, pigeons, and guinea-pigs by the intravenous injection of pure cultures of this organism.

Harbitz and Scheele were unable to confirm the findings of their confrère and state in this particular:—

> We believe it at present to be impossible to speak definitely on the question of the specificity of the organism demonstrated by Geirsvold, the more so *as Geirsvold himself makes certain reservations.* (Italics the editors'.)

There was, however, no lack of confirmation among investigators of other countries. Pasteur, Fullerton and MacCormac in 1908 "identified a micrococcus in the spinal fluid of patients with symptoms of poliomyelitis; inoculating this fluid in the subdural space of rabbits induced an ascending motor paralysis, with the recovery of the same micro-organism from the spinal fluid; the organism refused to grow on artificial media."

Schultze found a micro-organism present in the cerebrospinal fluid during the acute stage of an attack, and also on the thirteenth day of an attack; this organism was a diplococci, and cultivation was negative.

The bacteriological work of Wickmann, of Sweden, and of many German investigators, including Krause and Meinicke, Eichelberg, Leiner and Wieser, and Romer, was negative, while Potpeschnigg states that he found a Gram-positive diplococci in the cerebrospinal fluid of 14 cases of acute poliomyelitis. Shidler, of Nebraska, in 1909, reported that he made 14 lumbar punctures, and found a sterile fluid in 1 case only, and in that case the fluid was withdrawn four weeks after the temperature had subsided; in 9 cases a Gram-positive coccus was obtained, and in 3 a diplococcus; these micro-organisms were never found to be intracellular; cultures were inconclusive.

The results of the bacteriological research for the cause of poliomyelitis may be summarized as follows:—

Many independent investigators have demonstrated the presence of a micro-organism in the cerebrospinal fluid of cases in the acute stage of the disease; this organism is of a round, ovoid or kidney outline; it is susceptible to culture in a media of nutrient broth, which implies anaërobic environment; pure cultures of this organism produced experimental paralysis in animals; cerebrospinal fluid containing this organism produced experimental ascending paralysis in rabbits, which was capable of transmission to a second series of rabbits.

It was at this point that the bacteriologists seemed to be hung up; their results were not uniform; their pure cultures from the cerebrospinal fluid became sterile in from twelve to fourteen days; transmission was not carried beyond the second series in animal experimentation. They were agreed that they had at least a fleeting glimpse of an organism, "not quite like" anything they had hitherto studied. It is notable that one man, Beneke, of Marburg, whose work we will have occasion to refer to again later, demonstrated certain bodies resembling diplococci in sections of the pia and cord, obtained *post mortem* from cases of the acute disease. It was evident that the organism, whatever it was, did not belong to the group of readily cultivated and easily stained bacteria.

III. *Flexner's Filterable Virus of Poliomyelitis.*— Subsequent to the announcement by Flexner and Lewis that they had successfully transmitted epidemic poliomyelitis to the rhesus monkey, they published a study on the "Nature of the Virus of Epidemic Poliomyelitis" in the Journal of The American Medical Association, which is here given in part:—

"From the beginning our attention has been directed toward the solution of that fundamental question, the nature of the virus responsible for producing the disease, but the results of our studies

were until recently wholly of a negative nature. *We failed utterly to discover bacteria* whether in film preparation or in cultures that could account for the disease; and since among our long series of propagations of the virus in monkeys not one animal showed, in the lesions, the cocci described by some previous investigators, and we had failed to obtain any such bacteria from the human material studied by us, *we felt sure that they could be excluded from consideration.* We have, up to this time, made a very painstaking study of film preparations and sections taken from two specimens of human spinal cord, and many specimens of the spinal cord and brain and other viscera obtained from monkeys, prepared and stained in the most various ways, but *without finding* either *bacterial or protozoal parasites* that could account for the infection. The readiness with which epidemic poliomyelitis can be transmitted to monkeys and the failure to find visible and stainable parasites in the lesions of the spontaneous and experimental disease led to another line of investigation. It is known that the viruses of vaccinia and rabies, neither of which has been certainly demonstrated in films or sections of tissue, nor cultivated artificially, withstand very well the action of glycerin, while bacteria withstand it far less well.

"We therefore suspended in glycerin the comminuted spinal cords of monkeys affected with poliomyelitis, and after an interval of days we have inoculated the glycerinated virus into normal monkeys. Monkey No. 35 developed paralysis on the tenth day after inoculation, with characteristic lesions in the brain and cord. Monkey No. 58 was then injected with the cord of monkey No. 35 and developed paralysis on the eleventh day.

"The next series of experiments were planned to determine the probable size of the organism producing poliomyelitis, so far as this could be accomplished by the use of mechanical filters. . . . . Cord was triturated with sterile quartz sand, mixed with salt solution, thoroughly shaken and pressed through a Berkefeld filter. The clear and sterile filtrate was injected intracerebrally into Monkey No. 68, which developed paralysis on the seventh day following.

"From the foregoing experiments, taken in conjunction with the negative results of bacteriologic and histologic examinations, it would appear that the infecting agent of epidemic poliomyelitis belongs to the class of the minute and filterable viruses that have not thus far been demonstrated with certainty under the microscope."

In a later publication Flexner states:—

"The virus in aqueous suspension passes with readiness and little or no loss of potency through the pores of the densest and finest porcelain filters, namely, the so-called Chamberland filter. It passes with even greater ease through the somewhat less dense Berkefeld filter. It is extremely doubtful if the virus has actually been seen.

. . . .

"The filtrates are highly potent. Quantities as small as one one-thousandth to one one-hundredth of a cubic centimeter suffice to cause paralysis in monkeys after the usual incubation period when injected into the brain. The virus is highly resistant to external agencies and conditions. It withstands glycerination for weeks or months. It withstands drying over caustic potash for weeks or months without any or marked reduction in potency. It retains its virulence for weeks on being kept frozen at minus 2° to 4° C. On the other hand it is readily injured by heating, since temperatures of 45° to 50° C. maintained for half an hour render the filtrate incapable of causing paralysis. That the virus is a living organism must be concluded from the fact that such minute quantities of it suffice to carry infection through an indefinite series of animals."

The filterable virus theory of Flexner's, whose probabilities rested on the foregoing demonstrations,

(*a*) No visible or stainable bacterial nor protozoal parasites.

(*b*) Infective virus said to have passed with readiness through the densest and finest porcelain filters,

was greatly nullified by the fact that

(*a*) Many investigators found organisms, similar in their differences from all known bacteria, freely present in the spinal fluid drawn from poliomyelitis cases.

(*b*) Pure cultures of these organisms induced experimental paralysis in various animals.

(*c*) The virus was *not filterable* through Reichel filters. (Leiner and Wiesner.)

IV. *Dixon's Protozoön.*—The State of Pennsylvania during the summer of 1910 suffered from an extensive

epidemic of poliomyelitis of 1000 reported cases. Drs. Dixon, Fox and Rucker, of the State Department of Health, began a series of independent experiments which were carried on for some months, and the results published as a State Laboratory report March 2, 1911. This valuable report is given in part:—

"In examining the blood from acute cases of poliomyelitis (human and monkey) an organism was found differing in morphological characteristics from any heretofore described. The bloods examined were taken from 10 cases of acute poliomyelitis in children, and during the acute stage from 13 monkeys in which the disease had been artificially produced. *Defibrinated blood,* three weeks to two months old, *showed the organisms in increased numbers.* Cultures of blood from paralyzed monkey in blood-bouillon, plain bouillon, and blood-agar, examined after having been inoculated three weeks, showed the forms in increased numbers. *Filtered virus showed none of these organisms.* Blood-smears from 3 normal human beings gave negative results. Smears from the blood of 13 normal monkeys gave negative results; after inoculation with the virus the blood of these monkeys gave positive results. Blood-smears being fixed and stained, the organism was seen to be free in the serum as well as in the body of the red blood-cell. Stained with carbol-thionin the organism appeared faintly colored with a finely granular protoplasm; size, ten microns long by about eight microns wide; shape, curved at an angle of sixty degrees, with the curved end at times bulbous. The stain with iodine and sulfuric acid for cellulose was negative. Search for moving organisms in the fresh blood, defibrinated blood, and blood-bouillon was also negative."

These experiments suggested to the workers that the organism with which they were dealing was a protozoön, the conclusion at which Knoepfelmacher, of Germany, had arrived during experimental work with the organism. Beneke, of Marburg, while examining hanging-drop preparations of infected blood, on two occasions found small, *rapidly moving* bodies. Beneke also demonstrated Gram-positive bodies in the pia and sections of the cord, removed *post mortem* from human cases of poliomyelitis. There

is one more point in Beneke's experimental work which is suggestive; he succeeded in infecting 3 rabbits with poliomyelitis by introducing a solid portion of fresh, infected cord intradurally in 1 rabbit, and in the abdomen of the 2 remaining; the third rabbit developed convulsive movements and paralysis; they all died; and although no changes could be demonstrated in the brain or spinal cord of any of the animals, there was much hemosiderin deposited in the spleen of each.

Three points in Beneke's work lend strong support to the Dixon protozoön theory:—

(*a*) The demonstration of rapidly moving bodies in the infected blood.

(*b*) The demonstration of organisms in the section of pia and cord.

(*c*) The demonstration of hemosiderin in the spleen of three infected rabbits.

Protozoa.—Protozoa are one-celled animals which have crossed the border line dividing the viable from the vegetable world. All forms of bacteria remain in the vegetable kingdom.

There are several divisions of protozoa classified according to their morphology and other characteristics: (1) Rhizopoda, named for their means of locomotion, of which the ameba is the most familiar example. (2) The flagellates, including the trypanosomes and spirochetes, whose movements are undulating and whiplike. (3) Infusoria, interesting historically in that Jennings first found in one of their number "what must be considered the beginnings of intelligence, and of many other qualities found in the higher animals." (4) Sporozoa, the spore-forming protozoa, of which Doane writes: "No other group of animals is being studied more today by both physicians and biologists."

The trypanosomes are causative factors of sleeping sickness, and the epidemic paralysis of domestic animals in Asia which is known as surra, as well as other epidemic diseases. The spirochetes are of especial interest to the student of poliomyelitis, as it was Noguchi's discovery of the proper media for the culture of *Spirochæta pallida* which has enabled him to (January, 1913) present to the scientific world pure cultures of the organism of acute poliomyelitis. The organism of syphilis was grown successfully in pure culture only when sterile (living) tissue was added to the ascitic culture media, and similarly a pure culture of the poliomyelitis organism was produced. There has been some discussion as to whether the spirochetes *are* true protozoa, but Nuttall has shown that they should be classed on the animal side of the line.

Sporozoa.—Protozoa which are capable of taking on a resting stage during which the encysted organism develops great resistance to destructive agencies are known as spore-forming. Sporozoa are motile during the active stage; they may develop flagellæ or propel themselves about by means of pseudopodia; they are not dependent on the blood-stream for locomotion, but migrate through the body tissues as do the white blood-corpuscles. The polymorphous character of protozoa is well shown in Fantham's study of a small flagellate protozoön (*Herpetomonas pediculi*) which inhabits the alimentary tract of the louse. Fantham presents drawings of 28 modifications in form of this one organism; the major changes are from an oval, pre-flagellate form to an elongated flagellate form, and post-flagellate, small, oval and encysted resting stage.

Means of Transmission of Protozoa Outside of the Body.—It is now recognized that various members of the protozoön family are the occasion of most of the infectious

transmissible diseases of man, that the source of the infection is commonly inoculation, and that inoculation is commonly produced by a blood-sucking insect. The Director of the Lister Institute of Preventive Medicine, Dr. C. J. Martin, recently stated:—

> Spirochetes are such obligatory parasites that the agency of an insect transmitter seems almost necessary. The only certain way of producing the disease (relapsing fever) is by inoculating a minute quantity of the blood of a patient during the febrile stage. (Martin, "Insect Porters of Infections," British Medical Journal, Jan. 4 and 11, 1913.)

Epidemiological Endorsement of Dixon's Protozoön Theory.—It is a conspicuous fact that most of the protozoa which have been identified with disease were found in the tropics or semitropics, and proved to be the exciting agent of some so-called tropical disease. The plasmodium of malaria cannot be considered as an exception, because its most virulent forms are found to exist in tropical regions. The paralysis which is epidemic among domestic animals in North America coincidently with epidemics of human poliomyelitis, is clinically related to an epidemic paralysis which is very destructive to domestic animals in tropical Asia which is known as surra. A similar disease called nagana decimates the domestic animals of tropical Africa. These tropical epidemic paralyses are known to be occasioned by a trypanosome transmitted by biting flies. Should one of these diseases of domestic animals at the tropics extend its range to domestic animals and man in the temperate zone, marching to the north in step with summer, it would probably make a heavy invasion during the hot weather, and check rapidly as the oncoming of frost destroyed its customary environment. The tropical disease might remain an endemic infection in insect or human carriers, dormant until the return of

summer restored activity and virulence to its exciting organism. A study of many epidemics demonstrates that in just this manner poliomyelitis is manifested. Poliomyelitis, which is sporadic in winter in the temperate zone, becomes epidemic in the early summer and increases to maximum occurrence in the month of August. The most severe epidemics known to occur appeared simultaneously with seasons of unusual and prolonged heat and drouth in Scandinavia, North America, and England, and lessened with the cessation of extreme heat, a considerable period of time before natural agencies (frost and cold rains) checked the impetus of insect fecundity.

THE PLEOMORPHIC SPIROCHETE OF RABIES.

The clinical analogy of epidemic poliomyelitis to epidemic rabies is striking. The clinician notes the strong analogy both in symptoms and progress of paralysis, and death occasioned by paralysis of respiration, which occurs in both poliomyelitis and rabies. The Pasteur treatment is not infrequently followed by a temporary paralysis, and Landry's paralysis has been a sequel of this serum treatment.

Proescher, of Pittsburg, who has given four years' time to the demonstration of micro-organisms in rabies, announced the definite etiologic factor of this disease, January, 1913, as follows:—

Proescher was convinced that rabic virus must be microscopically visible because (*a*) he had found it nonfilterable, and (*b*) there was no diminution in the virus of a rabic dog which had been buried eight months. He was able by careful technique to demonstrate organisms in smears from the following sources:—

(*a*) A smear from the brain of an infected rabic rabbit killed in the paralytic stage.

(*b*) Smears from the brains of 12 dogs infected on the street, whose rabic state was demonstrated by transmission to other animals.

(*c*) Smears from the salivary gland and nerve-ganglion of a cow (proved rabic by transmission of infection to other animals).

(*d*) Smears from the salivary glands of 3 dogs (proved rabic by transmission to laboratory animals).

(*e*) From fixed virus obtained from four Pasteur Institutes in America (U. S. Marine Hospital, Washington, D. C.; Chicago; New York; Alexander's).

Smears and sections stained with a 1 per cent. carbo-azur-carbonate (Unna-Giemsa) demonstrated the "entire cycle" of rabies virus, cocci and bacilliforms, and spirochetes. These pleomorphic germs showed an achromatophilia to all aniline stains except the azur-carbonate. Proescher considers that the pleomorphism is satisfactorily interpreted as developmental forms of one organism, the resting, active, motile and spore stages. The bacilliforms were bipolar, one-half to one micron in length and one-third micron thick, isolated, or arranged in chains and groups; besides these small bacilliforms, there were larger, oval or round forms. It was remarkable that often a larger form was found at one end of the chain, and the succeeding ones gradually diminished in size to a very minute form. Proescher, noting that the biologic properties of rabies were akin to those of poliomyelitis, made smears from the cervical cord of a case of poliomyelitis; stained with azur-carbonate the smear showed a great number of coccilike forms, although the culture taken from the same media two hours after death remained absolutely sterile.

In these researches we again have evidence of the protozoan origin of a disease closely allied to or perhaps

only a modified form of acute poliomyelitis in the human being. A certain degree of intelligence seems to be exhibited by motile protozoa which congregate in the salivary passages of blood-sucking insects, from which point they most readily gain access to the blood-stream of the obligate host. Such intelligence or response to stimuli would indicate that the organism belonged in the class of quickened, protoplasmic, motile, one-celled animals, grouped as protozoa, rather than among the bacteria.

SUCCESSFUL CULTIVATION OF THE PLEOMORPHIC PROTOZOAN OF POLIOMYELITIS.

Proescher's suggestive announcement of the identification of the pleomorphic spirochete of rabies, January 4, 1913, was followed in February by the announcement that Noguchi, making use of the technique by him developed in the growth of the *Spirochæta pallida*, obtained pure cultures of the organism of poliomyelitis.

Noguchi's technique is given in his standard of identification of *Spirochæta pallida:* (*a*) presence of sterile fresh tissue in culture media; (*b*) strict anaërobiosis.

In a solid medium consisting of 1 part of ascitic fluid and 2 parts of weakly alkaline agar containing a piece of sterile fresh tissue, the *Spirochæta pallida* grew slowly and steadily around the tissue and formed faint, diffuse colonies.

The report of Noguchi's cultivation and identification of the organism of poliomyelitis follows:—

> The cultivations have been conducted both with Berkefeld filtrates (of poliomyelitis emulsion) and (poliomyelitic) tissues in substance. The culture mediums consist (first) of sterile, unfiltered ascitic fluid or of brain extract to which fragments of sterile rabbit kidney and a layer of paraffin oil have been added, and of these plus 2 per cent. nutrient agar-agar in proportions of 1 to 2. The first

mediums permit of a slow growth not visible to the naked eye, while the second (which are unsuitable for obtaining the initial growth) yield, after several days, visible minute colonies clouding the tubes. The cultivations are conducted under anaërobic conditions, and the colonies do not ascend to the summit of the deep layer of solid medium.

The minute colonies are composed of globular or globoid bodies, averaging in young cultures 0.15 to 0.3 micron in size. The bodies appear in a variety of arrangements: single, double, short chains and masses. Often they appear embedded in a material of different refractive index. In older cultures certain bizarre forms have been noted. The cultivated bodies stain a pale reddish violet in Giemsa's solution, and bodies of identical appearance have been demonstrated by Noguchi, also with Giemsa's solution, by a specially devised method in films prepared directly from the nervous tissues.

Monkeys have been inoculated with the cultures. Two series of inoculations have been made and a third series is in progress. Cultures from human tissues in the third and from monkey tissues in the fifth generation have caused typical experimental poliomyelitis in the monkey. The findings at autopsy and the microscopic appearances of sections of the spinal cord, medulla and intervertebral ganglia were characteristic of the disease. From the nervous tissues of these animals other monkeys were successfully inoculated and pure cultures recovered. (Jour. Amer. Med. Assoc., Feb. 1, 1913.)

It will be noted that Noguchi found the organism of poliomyelitis taken from young cultures globose in outline and very minute (less than half of one micron in size); while in older cultures the bizarre forms recorded suggest at once the entire cycle of developmental change in morphology of the rabic organism demonstrated by Proescher. In addition the cultivated globose bodies and similar organisms in films prepared from the spinal cord, medulla, and intervertebral ganglia were successfully dyed by the azur-carbonate (Giemsa) stain used by Proescher to demonstrate the rabic organism.

Noguchi's work confirms (1) Giersvold, who first successfully cultivated the organism of poliomyelitis; (2)

Beneke, who cultivated it in living anaërobic tissues by inclosing bits of infected cord in the peritoneum of a living rabbit; (3) Proescher, who demonstrated organisms in tissue of cervical cord from a case of poliomyelitis by staining with the Giemsa azur-carbonate; and many other investigators of Europe and North America.

CHARACTERISTICS OF THE ORGANISM OF EPIDEMIC POLIOMYELITIS.

From the foregoing it may be deduced that the etiologic factor of acute poliomyelitis is a pleomorphic, motile, anaërobic, pathogenic, obligative hemoprotozoön; with a developmental cycle consisting of a resting, motile, dividing, and resistant spore stage; which elaborates a virus having a destructive affinity for myelin and susceptible to great augmentation and modification; capable of pure culture in an anaërobic preparation of a solid or fluid, sterile, living body tissue; reacting specifically to the azur-carbonate dye; bearing a strong clinical and histologic analogy to rabies; pathogenic to man and domestic animals; endemic in the tropics; epidemic in the tropics and in the temperate zone during the summer season; pandemic under conditions of prolonged heat and drouth and other unknown factors most favorable for its wide diffusion.

EXPERIMENTAL TRANSMISSION TO MONKEY.

Acute poliomyelitis was first successfully transmitted to monkeys by Landsteiner and Popper, in 1908, by intraperitoneal inoculation of an emulsion of the spinal cord of a boy who died of poliomyelitis. They failed to transmit the disease from the first to a second series of monkeys at that time. In October, 1909, Drs. Flexner and Lewis produced the experimental disease in monkeys by intracerebral inoculation of a triturate of the spinal cord of 2

victims of the disease. They successfully retransmitted the disease to successive relays of monkeys, until a series of 25 had been produced. In April, 1910, by the courtesy of Dr. Flexner, Director of Rockefeller Research Laboratory, the writer was shown this epoch-making line of investigation. In numerous cages, lining a large and airy room, were monkeys, each of which presented one or more of those deforming disabilities previously seen to develop among children and adults of North-central Wisconsin.

This sight was one of the rare gratifications of life.

Fig. 5.—Experimental paralysis of all four extremities in monkey. (After *Romer.*)

This will be understood by every doctor in an invaded community, who, convinced that he was facing an epidemic of a communicable disease, had yet no authoritative proof to offer to indifference, prejudice, and cupidity that the disease was infective and transmissible, and demanded urgent and immediate isolation, improved sanitation, and other prophylaxis, to check its advance.

The analogy these apes presented to the cases in human beings was curious and startling. There were many paralyses of extremities, with the typical attendant atrophies and contractures. One had wrist-drop; another, a flail arm. There was a grotesque case of paralysis of the facial, with dropped jaw. One big male was paralyzed

from the flanks down, yet climbed about in his cage with surprising agility, dragging the atrophied limbs, less helpless than a child with paraplegia.

The original material used for these inoculations was obtained, *post mortem*, from 2 children who died during the acute stage, 1 on the third, and the other on the sixth

Fig. 6.—Experimental facial paralysis (right) in monkey. (After *Romer.*)

day succeeding the appearance of the paralysis. The virus from these cases was continuously transferred through series of monkeys, becoming more active during each successive inoculation. It could apparently be transferred indefinitely. Here was proof that infantile paralysis is an infectious disease, caused by a living organism capable of reproduction in the body of its host. The potency of a chemical toxin would be so diminished by re-

peated dilution and division as to be wholly inert, and a chemical substance has no power of multiplication nor reproduction.

The progress of the disease in the ape was a close analogue of the affection in a human being. The virus, a solution of the spinal cord of a victim, was usually in-

Fig. 7.—Experimental right oculomotor paralysis in monkey. (After *Romer.*)

troduced through a trephine opening into the brain-pan. When the ape recovered from the anesthesia, he appeared in every way normal for a period of time varying from four to thirty days. After a few days he was seen to be listless for a few hours, or with no marked symptoms of onset; he became ill with great suddenness. The paralysis appeared in from two hours to forty-eight hours following the period of incubation. The paralysis in more than one-half of the cases affected the lower extremities, and

otherwise varied in extent, location, and degree as it does in human beings. In some of the monkeys, lightly affected, there was motor weakness only, followed by recovery. In some cases the paralysis extended upward with bulbar involvement, paralysis of respiration, and death. The mortality rate constantly increased with an apparent

Fig. 8.—Experimental paralysis of right hind leg in monkey. (After *Romer.*)

increase of the virulence of the virus until it reached 100 per cent. of the animals inoculated.

The disease has been transmitted to monkeys by other investigators, and by other methods. Strauss and Huntoon, Leiner and Wieser, Romer, and Landsteiner and Leviditi, have all successfully transmitted the disease to apes. An emulsion of the infected cord has been successfully injected into the meninges, the nerves, the nerve-sheaths, and the peritoneum. The disease has been

caused by the artificial introduction into the stomach and intestine of some of the emulsion; this latter method was successful only when peristalsis was controlled by opium. The disease has been caused by rubbing an emulsion of the cord on the mucous membrane of the nasal passages after scarification of the same; by application to the intact

Fig. 9.—Experimental facial paralysis (right) in monkey. (After *Romer.*)

(?) nasal mucosa; by enforced inhalation of an emulsion of an infected cord, and by subcutaneous inoculation.

In the body tissues the virus has been demonstrated to be present in the blood and spinal fluid during the early stage or onset of disease. It has been found in the salivary, mesenteric, and other lymph-glands, and the spleen. It is supposed to be eliminated by the feces. Kling and Petterson demonstrated that the intestinal mucosa of a case was infective.

Poliomyelitis has been transmitted to monkeys by many experimenters; to rabbits, to guinea-pigs, and to pigeons. From infected rabbits the disease has been transmitted to other rabbits, and also to monkeys. Experimentation with other animals and fowls has so far failed to reproduce the disease. The failure to inoculate cats, dogs,

Fig. 10.—Experimental paralysis, facial and hypoglossal, in monkey. (After *Romer.*)

sheep, and horses with this disease is probably due to some lack of technique in the artificial inoculation, for evidence accumulates that many domestic animals are simultaneously attacked during an epidemic among human beings. (See Chapter I.) It may be due to an acquired immunity. When Pasteur wished to increase virulence in a pathogenic germ he inoculated a day-old guinea-pig. It is probable that the young laboratory animal would prove susceptible when the adult remained resistant.

METHOD OF TRANSMISSION OF THE MICRO-ORGANISM OF ACUTE POLIOMYELITIS (A) FROM ANIMAL TO MAN; (B) FROM MAN TO MAN.

("I eat and keep silence." A literal translation of the native name for an Egyptian blood-sucking fly.) There is but a single layer of epithelium between us and death, and the bite of an insect pierces the barrier.

Two years before the final identification of the specific micro-organism of poliomyelitis by Noguchi, several investigators, including Frost, of the Public Health Service; Mills and Dixon, of Pennsylvania, and Manning, of New York, suggested that the probable agent of transmission of this disease was a blood-sucking insect. The summer of 1912 Sheppard and Rosenau, of Massachusetts, confirmed this theory by the successful transmission of the disease from monkey to monkey by the biting stable-fly, *Stomoxys calcitrans*. Their work with the biting fly was immediately reconfirmed by Anderson and Frost, of the Public Health Service. The reports are given later. The agency of the stable-fly accounts satisfactorily for the transmission of epidemic paralysis from animal (especially the horse) to man. Manning, in the face of much opposition, announced and maintained that the bedbug, *Cimex lectularius*, was the logical and usual carrier of poliomyelitis from man to man. Van Gieson's (unpublished) experiments with the bedbug and poliomyelitis were all negative. Howard and Clark announced, in December, 1912, that bedbugs, seven days from feeding on poliomyelitic monkey, contained sufficient active virus to paralyze and cause death in a healthy monkey in which it was injected. It now seems probable that, in addition to the stable-fly and the bedbug, other blood-sucking insects may be implicated in the transmission of the disease.

Langhorst, of Illinois, has reported 2 cases apparently due to direct inoculation from the dog.

Inoculation.—The experimental production of poliomyelitis in animals has been achieved only by some method of inoculation. It has never been secured where a possible abrasion and therefore a wound surface could be absolutely ruled out. The routine method of experimentation has been the injection of an emulsion of infected tissues into the trephined brain of a monkey. Experimenters claim to have produced the disease by applying virus to the unabraded mucous surface of the nares, but if one delicate mucous cell was denuded in this process an inoculation atrium was thereby established.

As the successful production of experimental poliomyelitis depended on inoculation, it seemed reasonable to expect that spontaneous cases also arose from inoculation. Advocates of the contagion theory were at a loss to account for the fact that spontaneous transmission among laboratory monkeys was never known to occur, and the comparative rarity of multiple cases in families, secondary cases in institutions, and the lessened incidence of the disease in winter, the period when nasopharyngeal contagions multiply. Not only was experimental transmission of the disease dependent on inoculation, but the greatest incidence of the disease in epidemic form appeared during that season of the year when insect life reached the maximum. Observation of these facts led to the following investigations:—

STOMOXYS CALCITRANS.

(The Stable-fly. The Biting Fly. The Barn-fly. The Rain-fly.)

Owing to the frequent histories of insect bites of various kinds, it was deemed advisable that the entomological part of field work should be thoroughly investigated. There is a negative evidence

afforded by the failure of investigators to satisfactorily account for the spread of infantile paralysis through other channels of infection.

Reports.—Waltham, 2 cases, 1 in a very unsanitary house in the yard of which many stomoxys were seen ten days after the patient became paralyzed.

Twekesbury, State Infirmary, 6 cases. Usual domestic insects were observed, also an abundance of stomoxys, some of which were seen upon a screened enclosure in the grounds in which the children had frequently been placed before they were attacked.

Fig. 11.—The Stable-fly; Barn-fly; Rain-fly. (*Stomoxys calcitrans.*)

Woburn, 14 cases. Epidemic paralysis of pigs. Paralyzed cat. Stable-flies.

Somerville, 10 cases. Usual series of domestic insects, including stomoxys. In 1 case a history was obtained that one month before the attack in July the child was stung between the shoulders by a strange insect, about 8 o'clock in the evening, and at the same time an adult member of the family was bitten the same way. The sting was accompanied by a very sharp pain and was probably the bite of some species of tabanus (horse-fly), although it may have been stomoxys or some other biting insect.

Newton, 4 cases. Several cows lame in hind quarters. Biting flies.

Winchester, 2 cases. Stomoxys seen in abundance.

Pocasset, 2 cases. Usual domestic insects, including stomoxys and small horse-fly.

Hamilton, 1 case. Biting flies.

Marblehead, 1 case. An insect described as resembling a tick had been seen on the infant and had bitten it. Stomoxys had been killed in patient's bedroom.

Marlboro, 3 cases. Stomoxys abundant about premises and barnyard, also seen in house and bedrooms of patients.

Lowell, 14 cases. Paralyzed cat. Stomoxys.

Westford, 1 case. Mosquitoes and stomoxys.

Winchendon, 1 case. Houseflies and stomoxys.

New Bedford, 4 cases. Stomoxys.

Fall River, 13 cases. Stomoxys. One child had marks of bites on his body. An interesting case in an adult male, 73 years old, was seen, who gave a history of bites by stable-flies, followed in a week by febrile attack and paralysis of one arm. ("Biting Insects and Infantile Paralysis," Sheppard and Brues, Mass. Jour. of Economic Entomology, August, 1912.)

The stable-fly *Stomoxys calcitrans,* which is common to Europe and America, is closely related to the tse-tse flies both in structure and habits. It has been quite definitely associated with the spread of surra, and may reasonably be suspected of transmitting any disease represented by organisms in the blood. This fly has been found in the environs of practically every case of infantile paralysis examined by us with this end in view, and may quite possibly prove to be the insect responsible for its spread.

Many facts connected with the distribution of cases, together with histories of insect bites, suggest that the disease may be insect borne. Field work, together with the epidemiology of the disease so far as known, points strongly toward biting flies as possible carriers.

TRANSMISSION OF POLIOMYELITIS BY MEANS OF THE STABLE-FLY (STOMOXYS CALCITRANS).

By John F. Anderson, Director Hygienic Laboratory, and Wade H. Frost, Passed Assistant Surgeon, United States Public Health Service.

As a result of the thorough epidemiologic studies of poliomyelitis conducted by the Massachusetts State Board of Health from 1907 to 1912, under the direction of Dr. Mark W. Richardson, secretary of the board, evidence was collected which led the investigators to strongly suspect that the common stable-fly (*Stomoxys calcitrans*) played an important part in the spread of this disease.

At the joint session of sections I and V of the Fifteenth International Congress on Hygiene and Demography in Washington, Sept. 26, 1912, Dr. Milton J. Rosenau, of the Harvard Medical School, who has been working in conjunction with the Massachusetts State Board of Health, announced the result of an experiment which seemed to confirm most strikingly the inferences drawn from the epidemiologic work above mentioned.

Dr. Rosenau stated that he had infected several monkeys with poliomyelitis by intracerebral inoculation, exposed them daily—from the time of inoculation till death—to the bites of several hundred stomoxys, at the same time exposing 12 fresh monkeys to the bites of the same flies. At the time the announcement was made 6 of these 12 monkeys were reported as having developed symptoms characteristic of poliomyelitis, *i.e.*, illness followed by more or less extensive paralysis. Of these 6 monkeys 2 had died, 3 were paralyzed at that time, and 1 recovered after a brief illness. In the cord of one of the monkeys that had died were found the characteristic lesions of poliomyelitis, that is, perivascular infiltration and destruction of the motor cells of the anterior cornu. The cord of the other monkey was reported to have shown changes less characteristic of poliomyelitis, namely, degenerations of the motor cells without perivascular infiltration.

At the time of announcement a sufficient interval had not elapsed to determine the result of the attempt to transmit the infection to other monkeys by inoculation with the cord of one of the two that had died.

This experiment, giving an altogether new direction to the experimental study of poliomyelitis, appeared of sufficient importance to warrant an immediate attempt at confirmation.

In the experiment below reported it has been our object to repeat the conditions of that reported by Rosenau, and we are indebted to him for assistance and advice in the details of the experiment.

On October 3, rhesus No. 242 was inoculated intracerebrally with an emulsion of the cord of a monkey which had died of poliomyelitis. The virus used is a strain originally obtained from the Rockefeller Institute for Medical Research, kept at the hygienic laboratory for nearly two years, during which time it has been passed through a large series of monkeys.

Two hours after inoculation the infected monkey was exposed to the bites of about 300 stomoxys recently collected in Washington. Thereafter until death, on October 8, this animal was exposed daily for about two hours to the bites of the same flies, plus additional fresh stomoxys added from time to time as caught. This monkey (No. 242) developed characteristic complete paralysis on the afternoon of October 7 and died at 2 A.M. October 8.

Another monkey (rhesus No. 246), similarly inoculated on October 5, was then exposed daily to the bites of the same flies, beginning October 7. This monkey developed paralysis on the morning of October 9, soon becoming completely paralyzed and dying that afternoon.

Thus, from October 4 to October 9, inclusive, the flies used had access to two monkeys inoculated with poliomyelitis; first, rhesus No. 242, then rhesus No. 246. It may be noted that the incubation period in both these monkeys was very short—four days from inoculation to the development of paralysis.

Beginning October 4, two fresh monkeys (rhesus No. 243 and Java No. 241) were exposed daily for about two hours to the bites of these same flies; and beginning October 5 a third fresh monkey (rhesus No. 244) was similarly exposed. All three of these animals subsequently developed symptoms of poliomyelitis, as follows:—

Java No. 241 was found completely paralyzed on the morning of October 12 and died a few hours later. At autopsy tubercles were found in the lungs, liver, and spleen.

Rhesus No. 244 showed paralysis of the hind legs on the same day (October 12), but was, nevertheless, exposed again to the bites of the stomoxys from 10 A.M. till 2 P.M. At 3 P.M. the animal, being almost completely paralyzed, was chloroformed. At autopsy tubercles were found in the lungs, liver, and spleen, but apparently not sufficient to have been the cause of death.

Rhesus No. 243, which had appeared well on the morning of October 13, was found, at 4 o'clock that afternoon, to have a partial paralysis of the right hind leg. The following morning the hind legs and right fore leg were almost completely paralyzed. By 3.30 P.M. the neck also was paralyzed and the intercostal muscles somewhat affected. The animal was then chloroformed. At autopsy the internal organs appeared normal, except the spinal cord, which was edematous, the gray matter being congested. Sections of the cord, histologically examined, showed typical well-marked lesions of poliomyelitis; perivascular round-cell infiltration; foci of dense infiltration in the gray matter of the anterior horn; and destruction of some of the motor neurons.

The histologic examination of the cords of monkeys Nos. 241 and 244 has not yet been completed, but it is believed, on the clinical evidence, that they died of poliomyelitis.

To summarize: three monkeys exposed daily to the bites of several hundred stomoxys, which at the same time were allowed daily to bite two intracerebrally inoculated monkeys, developed quite typical symptoms of poliomyelitis eight, seven and nine days, respectively, from the date of their first exposure.

In order to confirm the diagnosis of poliomyelitis in rhesus No. 243, 1 cubic centimeter of an emulsion of the cord of this monkey was injected intracerebrally on October 14 into a healthy monkey (rhesus No. 250). This animal recovered promptly from the operation and remained apparently quite well till the morning of October 17, when a partial paralysis of the right fore leg was noted, progressing somewhat during the day. On the morning of October 18 both fore legs were completely paralyzed and the hind legs weak. In the afternoon of the same day the right hind leg was completely paralyzed, the left very weak, and the neck paralyzed. The monkey died at 10.30 P.M. and was immediately placed on ice until autopsy could be made at 9 A.M., October 19.

At the autopsy there was found some congestion of the lower lobe of both lungs, most marked on the left side, upon which the animal had been lying after paralysis developed. The meninges of the cord were markedly congested. On section, the cord appeared edematous, and the gray matter congested, showing minute hemorrhages. The site of inoculation appeared normal except for a slight clot. Cultures from this site have shown no growth. The other organs were normal in appearance.

Histologic examination of the cord showed lesions characteristic of poliomyelitis, intense congestion and perivascular infiltration, foci of round-cell infiltration here and there in the gray matter, destruction of the cells of the anterior cornu, and small hemorrhages in the anterior and posterior cornu.

Conclusion.

These results, in confirmation of those announced by Dr. Rosenau, would seem to demonstrate conclusively that poliomyelitis may be transmitted to monkeys through the agency of the stable-fly (*Stomoxys calcitrans*).

CIMEX LECTULARIUS.

(The Bedbug. Chinchbug. Wall Louse. B Flat. Mahogany Flat.)

At a certain stage of the study of the epidemic transmission of acute poliomyelitis the writer (Manning) found it compulsory to investigate the habits of the bedbug. It was found to be the accredited agent of transmission of a number of blood-borne diseases: typhoid fever (Dutton), tuberculosis (Rose), kala azar or dumdum fever (Patton), leprosy (Goodhue, Kerr), relapsing fever (Sergeius), infantile kala azar (Gabbi), bubonic plague (Verjbitski).

The most extensive and instructive investigation of the bedbug to be found in medical literature was that undertaken by Verjbitski, a Russ engaged in research in the Laboratory of the Imperial Institute of Experimental Medicine at St. Petersburg, whose report was translated and published in the Journal of Hygiene. Verjbitski's researches related to the spread of bubonic plague, but their results are so amazing, that the epidemiologist endeavoring to trace the progression of any blood-borne disease, and the physician likewise engaged will feel well repaid for time spent on the brief précis appended:—

Verjbitski's results were definite, proving that bedbugs fed on animals dying of plague communicated the plague to guinea-pigs for five days afterward; fleas fed on animals dying of plague communicated the plague to other animals for three days. Verjbitski says in his report: "These experiments were conducted with guinea-pigs. The plague culture was enhanced in virulence by passing through several guinea-pigs. The bugs used were *Cimex lectularius,* which is the usual domestic parasite. The strong irritation occasioned by its bite is caused by the action of the saliva which is injected into the wound. A bug never inflicts but one bite, and does not leave the place until it has filled itself with blood. Its body under these conditions acquires an egg-shaped form. The bedbugs, in series of 50, were applied to guinea-pigs dying of plague, and to an area of skin under the thigh which had previously been shaved. The results definitely proved that the bedbug transmits plague, and that as an agent of such transmission the bedbug is to be more feared than the much-dreaded flea of man and animals."

Verjbitski's summary, under fifteen heads, of the results of the 60 recorded experiments is here given: "(1) All bedbugs and fleas which have sucked the blood of animals dying from plague contain plague microbes. (2) Bedbugs and fleas which have sucked the blood of animals suffering from plague contain plague microbes only when the bite is inflicted twelve to twenty-six hours before death, that is, during that period when the blood contains plague bacillus. (3) The vitality and virulence of the plague microbe are preserved in these insects. (4) The plague bacillus is found in bugs which are not starved, one to seven days; in bugs previously starved four months, they are found eight to nine days. (5) The number of plague bacilli increases the first few days. (6) The feces of the infected bug or flea contain virulent plague bacilli as long as they persist in the alimentary canal of the insect. (7) The more virulent the culture with which is inoculated the first animal on which the bug was fed, the more certainly the infection was conveyed by bites. (8) The local inflammatory reaction in animals which died from plague occasioned by bites of infected insects was very slight or absent. In the latter case one could only locate it by the situation of the primary bubo. (9) Infected bugs communicated disease to healthy animals five days; fleas, three days. (10) Not more than two animals were infected from the same bug. (11) Crushing of infected bugs *in situ* in process of biting occasioned, in the majority

of cases, the infection of healthy animals. (12) Injury to the skin occasioned by the bite of the bug or flea offers a channel through which the plague bacillus can easily enter the body and occasion deaths from plague. (13) Crushed infected bugs and their feces can infect small punctures of the skin caused by bites for a short time after infliction of bites. (14) On linen soiled by crushed bedbugs (or fleas) or their infected feces the plague bacillus can under favorable conditions remain alive and virulent for five months. (15) Chemical disinfectants do not in the ordinary course of application kill the plague bacillus in the infected bug or flea.

"In crushed infected bugs the plague bacillus preserved its morphologic characteristics during all the time it was found in the bodies of bugs. In the midst of a great mass of well-preserved blood-corpuscles could be seen an enormous quantity of plague bacilli in almost pure culture. Experiment XLII: The inner surface of the hind leg in 6 guinea-pigs was scratched three times with a fine needle and the crushed bug was rubbed over the scarification; all of the 6 pigs died of plague in forty-seven to sixty-nine hours. Pure cultures of plague bacilli were obtained from the following sources: (1) the crushed infected bugs on bits of linen—(*a*) dried thirty-five days at room temperature, (*b*) one hundred and thirty days in damp environment at 4 to 5° C., (*c*) exposed during eight days to direct sunlight, and (*d*) frozen ten days at 5° C. to 18° C.; and (2) from the feces of bugs which had been allowed to dry on linen at room temperature for ten days. These were all verified by the inoculation of guinea-pigs. From the results we must conclude that clothing and bedclothes which are soiled with material from infected insects, obtained either by crushing them or from their feces, can serve during a long time as a source of infection. The clothing of people who live in dirty, unhygienic surroundings is generally covered with spots from crushed bugs and their feces."

The following description of the bedbug is quoted from the bulletin, The Bedbug, of the U. S. Entomological Bureau:—

The bedbug has accompanied man wherever he has gone. Vessels are almost sure to be infested with it. It is not limited by cold. The presence of the bedbug in a house is not necessarily an indication of neglect, for, little as the idea may be relished, this insect may often

gain access in spite of all reasonable precautions. It is apt to get into the trunks and satchels of travelers. It migrates from one house to another, sometimes for a period of several months, gaining entrance daily. Migration is apt to take place if the inhabitants of an infested house leave it. With the failure of their usual source of food the bedbugs pass along walls, water-pipes and gutters, and gain entrance to adjoining houses. The bedbug is thoroughly nocturnal in habits and displays wariness, or intelligence, in its efforts at concealment during the day. It usually leaves the bed at the approach of daylight, to go into concealment in cracks in the bedstead, or behind wainscoting, or under loose wall-paper, manifesting its gre-

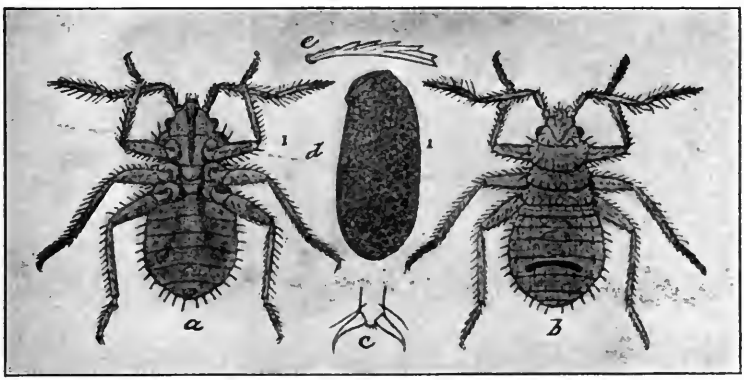


Fig. 12.—Bedbug (*Cimex lectularius*): Egg and newly hatched larva: *a*, larva from below; *b*, larva from above; *c*, claw; *d*, egg; *e*, hair or spine of larva. Greatly enlarged, natural size of larva and egg indicated by hair lines (original). (U. S. Circular No. 47, "The Bedbug.")

garious habit by collecting in masses. The inherited experience of many centuries of companionship with man has resulted in a knowledge of the habits of the human animal, and a facility of concealment, particularly as evidenced by its abandoning beds and often going to distant quarters for protection and hiding during daylight. The bedbug belongs to the order Hemiptera, characterized by possessing a piercing and sucking beak. The bedbug, though normally feeding on human blood, is able to get more or less sustenance from the juices of moistened wood, or the moisture in the accumulations of dust, etc., in crevices in flooring. The biting organs of the bedbug consist of a heavy underlip within which lie four thread-like,

hard filaments, which glide over each other with an alternating motion and pierce the flesh. The blood is drawn up through the beak, which is closely applied to the point of puncture, and the alternating motion of the setæ in the flesh cause the blood to flow more freely. In common with other insects which attack man, these pests may be the transmitters of contagious (?) diseases. The bite of the bedbug is poisonous to some individuals. To such the presence of the bugs is sufficient to cause the greatest uneasiness. With others, however, the presence of the bugs may not be recognized at all, and except

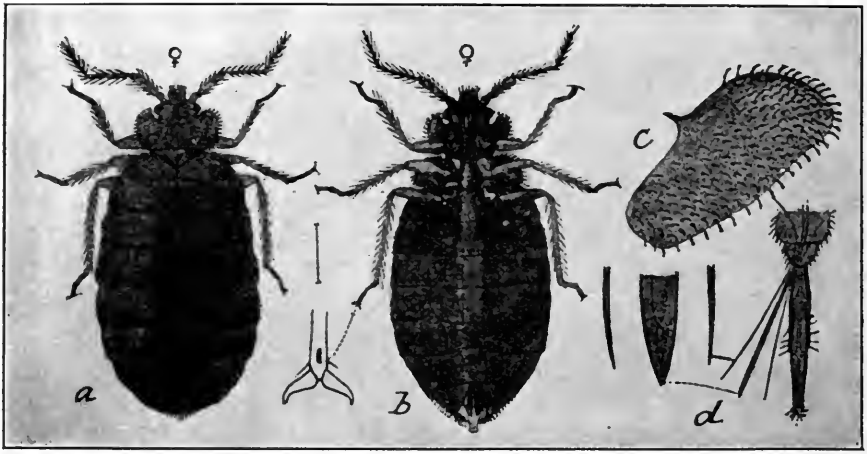


Fig. 13.—Bedbug (*Cimex lectularius*): *a*, adult female, gorged with blood; *b*, same from below; *c*, rudimentary wing pad; *d*, mouth parts. *a*, *b*, much enlarged; *c*, *d*, highly magnified (original). (U. S. Circular No. 47, "The Bedbug.")

for the occasional staining of the linen by a crushed individual their presence might be entirely overlooked. The bedbug is known to be able to survive for long periods without food. In unoccupied houses it can undoubtedly undergo fasts of extreme length. Individuals obtained from eggs have been kept in sealed vials in this office for several months, remaining active, in spite of the fact that they had never taken any nourishment whatever. Bedbugs are said to lay several batches of eggs during the season and are extremely prolific. The eggs are white, oval objects, and are laid in batches of one-half dozen to fifty in cracks and crevices where the bugs go for concealment. The eggs hatch in a week or ten days. Breeding experi-

ments conducted at this office indicate seven weeks as the period from egg to adult insect.

Are bedbugs a common factor in American homes? In each of the following instances I have observed bedbugs in large numbers: (1) Having had occasion to trace the source from which bedbugs were found on a white infant in a North Carolina home, I investigated the sleeping quarters of the colored cook and maid; the bed-frame under the mattress in each bed was a crawling, seething mass of bedbugs. (2) Tracing to their source the bed-

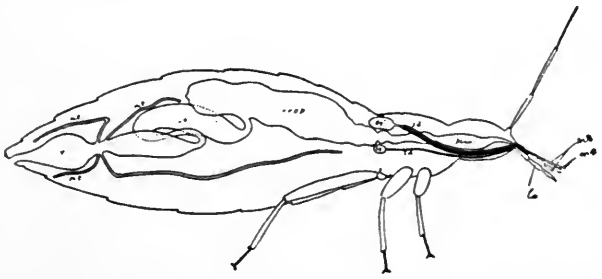


Fig. 14.—Alimentary canal and mouth parts of bedbug (*Cimex lectularius*). ("Insect Porters of Bacterial Diseases," *Dr. C. J. Martin,* Director Lister Institute Preventive Medicine.)

bugs which appeared in a scrupulously kept medical ward of Cook County Hospital, Chicago, I found the bed of the kitchen man, which he had previously made and "bugged" himself, alive with bedbugs. The bed-frame, drawn out on the concrete floor, was swabbed with alcohol and then fired, when the bedbugs literally boiled out of every joint in the iron. (3) Attending at night an obstetric case in a tenement while externe at the Chicago Lying-in Dispensary, I found hundreds of these crawlers descending the board partition which formed one side of the room. Called to the apartment of an unknown woman at night, I found her bedclothing swarming with bedbugs. Composing myself to sleep in a richly upholstered reclining chair

on a train in the Southwest in late October I was at once attacked by bedbugs in such numbers that all portions of the body felt as if scorched with flame.

Observation demonstrates that constant distribution of the bedbug among members of the various social classes takes place: the physician returns from the slum case and the lawyer from the court where bedbugs swarm; the maid takes her half-day in a tenement home, the daily paper is distributed by a tenement dweller, the hand laundry often returns from a tenement district; the vacation is spent in unfumigated summer camps, and the traveler's bag or trunk is a usual hiding place for cimex; men, women, and children of all social classes come in close contact in railroad stations, transit lines, theaters, schools, moving picture entertainments, summer amusements, and public inns. The invasion of the American home is more successfully accomplished by the gentle art of concealment practised by the retiring but ubiquitous bedbug.

Such instances as the following are difficult to explain by any theory of personal contact, direct or indirect:—

"In a sporting camp in a northern forest the 5-year-old son of the proprietor had been in camp since early spring. He had never left the camp during the summer. The camp was five miles from any other camp or house and ten miles from the frontier town. There had been no other children in camp during the summer and no illness of any kind in the few guests or among animals. The few guests who were there came from the large cities, put on their hunting and fishing clothes, and lived out of doors. In October the child developed a typical infantile paralysis which still persists in one leg." (Lovett and Richardson.)

The foregoing occurrence is readily explained by the assumption that an infected bedbug found its way to a guest's garments while en route, and later attacked the boy.

GEOGRAPHIC RANGE OF CIMEX.

Acute poliomyelitis has been reported in epidemic form from every continent during the past decade: Europe, Asia, Africa, Australia, North and South America. It therefore seemed well to ascertain the range of the insect suspected of transporting the disease. English scientists in England and India term it the Indo-European bedbug; Verjbitski states that it is commonly found in the homes of the insanitary Russian peasant; L. Lodian, an American engineer employed on the construction of the Siberian Railroad, wrote: "I tell you, the Siberian gentry are almost as large as our roaches, and their bite leaves an inflamed poison wheal about one-half the size of a hazelnut. Experienced travelers often prefer to lay their rugs on the floor and sleep there, taking the precaution, however, to empty the contents of a kerosene lamp on the floor in a sort of sanitary cordon; inside that ring they can sleep with some degree of immunity from the bedbugs." Miss Underwood, a returned missionary, states that while in China, after a brief daytime nap, the gown might be mottled with bedbugs. Bedbugs are found in Australia. In Africa the danger of association with cimex is so well known by the common people that caravan tents are known as "bugwalks," and avoided by men hiking to the diamond fields. The bedbug is a Pan-American pest.

BURDEN OF PROOF THAT CIMEX CARRIES POLIOMYELITIS.

It has been said that any insect, to merit consideration as an obligatory factor in the transmission of poliomyelitis, must be of (1) worldwide distribution (as poliomyelitis is pandemic), (2) perennial prevalence (as cases occur throughout year), and (3) capable of wide migrations

(as often there is a wide jump between cases), to which we would add that (4) its distribution must be urban as well as rural (as many cases in city as country), that (5) it must show a marked numerical increase in summer (with maximum incidence of cases), and that (6) *its preferred food should be the blood of man.*

1. That cimex is of worldwide distribution may be seen by reference to foregoing paragraph on geographic range.

2. Perennial prevalence. Cimex was formerly a hibernating insect, as may be deduced from the fact, demonstrated by the United States Bureau of Entomology, that the insect could endure fasts of many months' duration, sealed in glass tubes, and emerge alive and vigorous. Modern methods of artificial heating of homes in winter, both of the rich and poor, and domestication in these habitations have rendered hibernation unnecessary, for shelter and food are obtainable throughout the year. In January, 1912, the writer observed an adult bedbug migrating from one passenger to another in the brilliantly electrified coach of a Hudson tube. This migration was taking place in midwinter.

3. When Flexner placed stress on the factor that an insect, to convey poliomyelitis, must be capable of wide migration, winged insects were doubtless in mind. With modern transportation methods, an insect parasitic to man and with the art of concealment in clothes, etc., could be carried to distances beyond the power of the winged insect. This is the case with the bedbug.

4. The insect must have an equal distribution in city and country. Many cases of poliomyelitis have occurred in city homes which were remote from stables, greatly lessening the probability that the child had been exposed to the bite of the stable-fly. Some statistics have been

published and an impression has gone abroad that poliomyelitis is a disease of the country rather than of the town. Our experience in a city clinic inclines us to the belief that this statement is based on insufficient data. Sheppard, of Massachusetts, states that the 1911 epidemic focused in the city of Springfield, Mass.

Bedbugs abound in city tenements. The cleanly tenant is at the mercy of his uncleanly neighbor, and they are both wholly dependent on the janitor, for the efforts of one family will never make a material reduction in the number of bedbugs in a building.

5. Marked numerical increase in summer. The maximum incidence of poliomyelitis occurs during July and August in the north temperate zone, coincidental with the maximum incidence of insects, including bedbugs. On inquiry the writer has been informed by several housekeepers that young, newly hatched, white bedbugs are seen only at this season in temperate zone.

6. Preferred food, the blood of man. The common blood-sucking parasites of man are the bedbug, louse, flea, and house mosquito. It is quite possible that any one of these insects may transmit the organism of poliomyelitis from man to man. As poliomyelitis occurs any month in the year, it is doubtful that the flea, mosquito, or stable-fly, which are all annual and seasonable and disappear at the oncoming of frost, could be the common carriers of the disease. The louse may also be excluded from consideration, as it is found only among that class of citizens whose members are grossly unclean.

COINCIDENTAL PRESENCE OF BEDBUGS AND POLIOMYELITIS.

It is a striking fact that in every epidemiologic investigation of acute poliomyelitis in which a careful inquiry

has been made regarding the presence of bedbugs, they have been found to be present. As this insect has carried the art of concealment to a high power, it is certain that they are present and unrecognized in still more cases than those reported.

The first epidemiologist to make vermin a matter of routine investigation during an outbreak of poliomyelitis was Lovett, of Massachusetts. During the extensive invasion of 1909, a particular investigation was made of 150 cases which occurred in 142 families. The blood-sucking insects found present are given in the following tables:—

BITING INSECTS PRESENT IN HOMES OF 150 CASES OF ACUTE POLIOMYELITIS.

Fleas were present in	2 homes.
Biting flies were present in	3 homes.
Bedbugs were present in	31 homes.
Mosquitoes were present in	75 homes.

(Massachusetts, 1909. Lovett, Bull. Mass. State Board of Health, June, 1910.)

BITING INSECTS PRESENT IN HOMES OF 200 CASES OF POLIOMYELITIS OCCURRING IN 185 FAMILIES.

Sand-fleas were present in	1 home.
Fleas were present in	2 homes.
Biting flies were present in	7 homes.
Bedbugs were present in	39 homes.
Mosquitoes were present in	67 homes.

(Massachusetts, 1910. Sheppard.)

Dr. Sheppard adds to this report: "It will be seen that biting insects in families were present in greater number than the history of bites; I am inclined to the belief that the truth as to bites has been in a large measure withheld." (Sheppard, Report submitted to Massachusetts State Board of Health, May 1, 1911.)

In an investigation of the home surroundings of 33 cases of acute poliomyelitis occurring in and near New York City during 1911, which were treated at the hospital of Rockefeller Institute, bedbugs were found present seven times in all. The indifference with which their presence is frequently regarded may be judged from the statement of one father that the "bedbugs were worse in the children's room." This may not be the apathy of indifference, but a result of total inability to cope with a problem which should be one of municipal control, rather than ineffectual individual effort. The two first cases quoted illustrate a point the writer desired to make clear, that this vermin (cimex) is found equally distributed in city and country:—

Case 5. John O., aged 21 months. Paralysis of both legs complete; left forearm weak. Wristdrop. Clawhand. Tenement house. Eleven other families in house. Only parasites noted are bedbugs. The father states that these are worse in the children's room.

Case 7. James C., aged 18 months. Complete flaccid paralysis of both legs. Right arm weak. Neck and back muscles weak. Habitat, four-family house in the country; clean, and has good air. The mother states that there "are lots of bedbugs, cockroaches, and chicken-lice."

Case 21. Helen N., aged 6 years. Right facial paralysis. Many water-bugs, bedbugs, and fleas in West Side home.

Case 32. Patrick T., aged 23 months. Dragged one leg. Habitat, second-floor-front flat. Father, mother, sister, with acute poliomyelitis, and patient slept in one room. Flies, mosquitoes, bedbugs, and roaches. (From "A Clinical Study of Acute Poliomyelitis," Peabody, Draper, and Dochez.)

A history of insect bites is not infrequently given in cases of poliomyelitis. In Sheppard's report of the Massachusetts epidemic of 1911, it is said that an "insect resembling a tick had been seen on the infant and had bitten it before the symptoms of poliomyelitis developed." The insect which "resembles" a tick most closely is the bedbug.

The following suggestive case was recently reported by Dr. Spiller, Professor of Neuropathology, University of Pennsylvania:—

In another case of epidemic poliomyelitis, seen with Dr. W. B. Stewart, of Atlantic City, a bite of an insect produced quite a severe sore on the foot, and this was followed in a few days by paralysis. Whether an etiologic relation can be attributed to this bite or not is uncertain. (Spiller, Diagnosis of Poliomyelitis, Penn. Med. Journal, December, 1911.)

Professor Spiller has most kindly provided more extended notes of this very interesting case as follows:—

The patient was a girl, 14 years of age. She lived in Atlantic City, over a store. Atlantic City is on an island. She had gone to the mainland on a picnic, and during the evening was bitten by some insect. The bite was on the foot, and within a few days the sore was sufficient for her to seek medical attention. The bite, as I recall, was on a part usually covered by a shoe, but probably she had on a low shoe. A few days after she was bitten she developed symptoms of poliomyelitis. The paralysis was extensive in all limbs. No case of poliomyelitis in Atlantic City or on the mainland was known at that time. I cannot attribute the poliomyelitis to the insect bite.

The history of this case is very suggestive. *Cimex lectularius* may be considered a permanent resident of tenements and insanitary lodging houses, and the floating population of Atlantic City doubtless fetch and carry many members of his family.

The possibility of direct transmission of poliomyelitis from the acute case to any person entering the patient's room by inoculation from a bedbug which has been feeding on the patient is apparent. Such transmission through unknown agency is often reported, and the following case is suggestive in this connection:—

New York Hospital for Deformities and Joint Diseases, Poliomyelitis Clinic. S., schoolgirl, aged 9 years; onset May 17th; two

days before onset visited a playmate ill with poliomyelitis, in bed and paralyzed; S. was reproved by her sister for eating food which was on the bed of the sick girl, and by her mother for the same reason when she returned home. Attack, high fever, twitching, symptoms of ptomaine poisoning, general spastic condition, succeeded by paralysis from hips down, with paresis of upper extremities.

The well-known habit of the bedbug of crawling from a bed to the clothing of any person coming in contact with the draperies would account for the transmission of this disease by a third party who was and remained a healthy carrier. The agency of cimex offers a possible solution of the transmission of poliomyelitis in this record, excerpted from Dr. John Armstrong's study of an epidemic of 17 cases in a small community in Minnesota:—

D. W., female, aged 5 years; onset August 6, 1909; August 7th, paralysis of right lower leg.

August 24th, this child was visited by her grandmother, who on August 26th went to visit another grandchild, residing in a town in which no poliomyelitis had developed. A third grandchild was temporarily at the home of the second. The third child became ill, developing a paralysis August 28th. On August 30th the second grandchild became ill and also developed a paralysis. Is it possible (Dr. Armstrong inquires) the grandmother carried the infection and transmitted it to the second and third child? (Armstrong, Poliomyelitis, Pediatrics, August, 1910.)

It would be difficult to prove the migration of cimex from the sick child to the well grandmother, but that such migration takes place I have recently had proof:—

The evening of January 28, 1912, returning from Atlantic City on the Pennsylvania Railroad, we transferred to the Hudson tube at Manhattan Transfer. The coaches are the last word in safe and sanitary construction, being made throughout of steel and concrete. Seats extend lengthwise of the coach. Across from our party sat a small family group of the immigrant class. The mother,

a robust creature of 20 years, held a small, puny infant, in marked contrast to her own buxom appearance. The man, a well-built young male, sat limp and pallid, with his emaciated hands crossed feebly on his overcoat. There were several bags disposed about them, and one behind the man's feet.

While speculating as to the probable illness which had so depleted this young foreigner, my eye was caught by an insect which crawled out on the floor from under this bag. It was a fair-sized bedbug. It advanced across the aisle several inches and returned as fast to shelter when some one passed down the aisle. The coaches are brilliantly electrified and I had no difficulty in watching the maneuvers of this pest. Three times the bug essayed crossing the aisle, and each time returned to the shelter of the bag, as people entered the coach at stations. The fourth trip cimex came directly across the aisle; when within three inches of the clothing of two ladies, I crushed him with the toe of my shoe; the blood with which he was distended made a mark on the smooth concrete floor three-fourths of an inch in length.

The two notable facts of this migration are:—

1. Cimex was definitely migrating from sick to well.
2. This migration was taking place in midwinter.

If the premise is granted that cimex may be the agency of transmission of poliomyelitis, we would expect to find the disease endemic in certain houses. Wickmann noted such apparent endemicity, and reported it as proof of the contagious nature of poliomyelitis:—

> The disease was not generally spread through the city (Stockholm), but was particularly localized in certain parts, so that in neighboring houses groups of cases of 3, 5, or 7 occurred. In one instance there occurred a case in one dwelling house from which the family removed on October 1st. A second case developed in this

same house not long after the entrance of the family that moved into the rooms vacated by the first family. (Wickmann.)

If cimex proves to be the usual agent in the transmission of poliomyelitis, there will be explained the reason for non-development of secondary cases of the disease in the well-ordered hospital or ward. The modern hospital, with fumigation and removal of patients' clothing, and frequent fumigation of wards and rooms, does not harbor this pest. The unclean hospital ward which harbored cimex might, then, be responsible for secondary cases occurring in institutions.

Cases of poliomyelitis which develop during confinement in a jail (Harbitz, of Norway; Manning), and secondary cases occurring in institutions (Tewksbury, Sheppard), and also cases developing in a hospital (Harbitz) strongly suggest that transmission of the disease and inoculation have been produced by a blood-sucking insect. If these institutions are not subjected to a semi-annual fumigation, the possibility is at once aligned in the class of probabilities. As we have seen, the bedbug is the insect adapted by habits of concealment, and feeding, to be the guilty party.

Experimental transmission of acute poliomyelitis by the agency of the bedbug, from a poliomyelitic monkey to a healthy monkey which became paralyzed and subsequently died, was reported in December, 1912, by Howard and Clark, of Rockefeller Institute. The organism remained active seven days after feeding. Bedbugs are extremely resistant to destructive measures, and the human being has but a single layer of epithelium for protection from the inoculation of disease which may follow their bite. That bedbugs may transmit acute poliomyelitis has been demonstrated; they may prove to be the usual and common carriers of the disease from man to man.

CONTACT TRANSMISSION OF ACUTE POLIOMYELITIS.

The theory of contact transmission of poliomyelitis through secretions from the mouth and nose, which was advocated by Flexner, has had wide acceptation by the public, and, unfortunately, from patent medicine vendors, who have advertised and distributed, gratis, samples of "cures" for infantile paralysis, with a formula based on nasal antisepsis.

Many investigators have proved that the nasal and buccal secretions of the acute case, and the mucous membrane removed from these areas after the death of the subject, will transmit the disease when inoculated into the normal monkey.

Osgood and Lucas were able to transmit the disease from the tonsils and adenoid tissues of monkeys as late as five months and a half after the attack was over. They were also able to transmit the disease from the nasopharyngeal mucosa of a monkey that had been kept alive by careful nursing, and found the germs present at the end of five and a half months. They obtained the tonsils and adenoid tissue removed from a little girl six months and ten days after having had a spastic paralysis from this disease. The preparation from these was injected in various amounts into three monkeys. One of these animals developed typical signs of the disease accompanied with paralysis.

Kling and Petterson demonstrated the presence of the active virus in the secretions of the mouth, the nose, the trachea, and in the intestines after death from this disease. They also have found it in the mouth and pharynx and in the intestinal canal of patients during life. They obtained the secretions of the mouth and pharynx

by thorough rinsing with a syringe. The intestinal mucus was obtained after evacuating the intestines by means of an enema, and then thoroughly washing out the sigmoid with salt solution. The fluids thus obtained were filtered and *injected* into the peritoneal cavity and into the sciatic nerves of monkeys. In all, 13 patients were examined and the presence of the virus demonstrated in 12 by the production of typical paralysis in the monkeys.

Transmission by means of the nasal, buccal, and pharyngeal secretions when an inoculation atrium is present may not be of frequent occurrence, for Rosenau, Sheppard, and Amoss wholly failed in producing such transmission in a series of 18 cases. (Boston Medical and Surgical Journal, May 25, 1911.)

As we have previously stated, there seems to be reasonable doubt that transmission has been experimentally produced when a solution of continuity and therefore an inoculation wound could be absolutely ruled out, and yet the presence of active virus in or on the mucous membranes must receive due consideration in all measures which contemplate the prophylaxis or treatment of the disease.

TRANSMISSION OF ACUTE POLIOMYELITIS THROUGH THE DOG.

Epidemic paralysis among domestic animals has been found to occur coincidently with epidemic poliomyelitis by all epidemiologists of the disease. The subject is fully discussed in the first chapter of this volume. Paralysis of horses, dogs, and cats are most frequently noted. The association between master and brute is perhaps most intimate in the case of the dog. The fact that the mucous secretions or saliva of cases of acute poliomyelitis have been shown to be infective has a strong bearing on the

following report by Langhorst, of Illinois, on the apparent transmission to 2 cases of acute poliomyelitis through the dog:—

Report of Cases.—Case 1. The patient, aged 35, was a man of good habits and athletic build. He was married and had one child. Previous history was negative, with the exception of what I shall mention later. He was taken with a severe cough and sore throat about one week before paralytic symptoms appeared. He continued his outdoor work of driving a wagon, which was arduous, at that time, on account of the extreme cold and snow. The symptoms developed rapidly; he felt weak in his limbs one afternoon, and that evening, after going to the toilet, he was unable to return without assistance. I saw him a few hours later and found him completely paralyzed. He was gasping for breath and was intensely cyanosed; his legs, arms, abdomen, chest, neck, and throat were paralyzed. He spoke with great effort and was unable to swallow; his eye muscles were intact and their reflexes were normal. All of his other reflexes were absent; sensation of heat and cold was normal; only slight pain was present on pressure over nerve-trunks; he could locate and differentiate between pin-pricks with the sharp and the blunt end. The sensorium was clear; the patient was in good spirits and had no sense of fear. Loud bubbling râles filled his chest and he was continuously making ineffectual efforts at coughing, to try to bring up the mucus that was choking him. His vesical and rectal sphincters were tonic. Pulse was 130, temperature 100°, respiration 45.

By compressing his thorax, and by fixing the diaphragm (using pressure over upper abdominal region), we were able to help him to expectorate considerable mucus of a frothy nature. At times the respirations were so labored and the cyanosis so urgent that artificial respiration was resorted to, and kept up for hours at a time. Dr. Peter Bassoe saw the case, in consultation, and he favored the diagnosis of acute anterior poliomyelitis, but also considered the possibility of its being Landry's paralysis. On account of the increasing dyspnea, Dr. Richter, who had been using an improved pulmotor (made from a vacuum cleaner), was called in, to use his apparatus. This was used with good results, and was alternated with the Sylvester method. Atropine, strychnine, caffeine, and camphor were used hypodermically; normal salt solution was used by rectum, by the drop method. The urine contained no albumin, and the examina-

tion of the sputum showed many leucocytes, and, among other pus organisms, Pfeiffer's bacillus. After the third day the patient was able to move his hands and legs slightly, but after five days of severe suffering he became delirious, and on the sixth day died.

From the history I learned that the patient's dog had been sick about two or three weeks before, and was unable to stand on its hind legs. The patient fed the dog and cared for him. The patient remembered that he had a few scratches on his hand, and that on one or two occasions, the dog licked his hand. Flexner has shown that the nasal mucosa is one of the points of egress of the virus. If the dog had been inoculated with the virus at some time and had not immediately contracted the disease, he may have been a carrier and not contracted the disease until his vitality was lowered by living in a kennel in the cold weather. The dog could have inoculated his master with the secretions from his nose and mouth.

Case 2. The patient, a boy of 6 years, was brought to my office Aug. 9, 1912, to be treated for a bite inflicted by his pet dog. I saw him about an hour after the injury and applied a 1 per cent. solution of formaldehyde and then used tincture of iodine. The wound healed nicely. The boy then accompanied his parents on a trip to Canada, leaving here August 23d. While in Canada, on the 30th, the boy had a little fever and complained of his neck being stiff. The family immediately returned to Elmhurst, arriving here September 1st. I saw the boy at this time; he had a slight fever (temperature 101°), and did not complain greatly. He had pain in the back of his neck and in his head, and he felt weak. His reflexes were present and he was able to stand and walk. When I saw him the next morning he was paralyzed in all of his extremities; the chest and neck muscles were also involved. The reflexes were absent. Dr. Abt saw the patient with me and called it infantile paralysis. Dr. D'Orsay Hecht also saw him a few weeks later and agreed to the diagnosis. The boy is gradually recovering. ("Possible Transmission of Poliomyelitis Through the Dog," Langhorst, Jour. Amer. Med. Assoc., Dec. 28, 1912.)

Langhorst considers that poliomyelitis is undoubtedly transmitted to man through more than one intermediary, and points out that the biting fly which attacks animals may transmit the infection to the dog, which thereupon contracts the disease. Langhorst's contribution is of

great interest and value, not only that it demonstrates a possible new carrier of epidemic poliomyelitis in close association with and therefore a direct menace to man, but also that this report is a new link in the chain of evidence slowly forging which unites poliomyelitis and rabies in a relationship which simulates identity. There is a close analogy between these two diseases; the toxin of each is widely diffused through the system, and localizes in a destructive attack on the central axis; the atrium of infection of each, so far as has been shown, is through a wound; Proescher, of Pittsburg, has demonstrated their close biological analogy, and now Langhorst has followed with the report of the transmission of a paralytic infection of the dog to the human adult, who developed a typical case of acute poliomyelitis of the ascending paralytic type, with death from asphyxia on the sixth day. The world of medicine is much indebted to this scientific observer.

To summarize the foregoing, the present status of knowledge of the method and agent of transmission of acute epidemic poliomyelitis is that:—

Poliomyelitis has been artificially transmitted to monkeys by:—

(*a*) Inoculation: I. With poliomyelitic tissue. II. With poliomyelitic secretions. III. With comminuted bodies of bedbugs which had previously fed on infected monkeys. IV. By the bites of stable-flies which had previously fed on infected monkeys.

Poliomyelitis has been spontaneously produced in man by:—

(*b*) Inoculation: I. From the saliva of a paralytic dog.

The theory of transmission of acute poliomyelitis by contact contagion or fomites remains unproven.

CHAPTER III.

Predisposing Causes.

CERTAIN factors may prove active in causing this infection by:—

(*a*) Increasing susceptibility of host.

(*b*) Facilitating transmission of infection.

(*c*) Increasing virulence of infectious virus.

Race.—No race is known to be exempt from this infection. The white, yellow, brown and black man have all been included in reports of epidemics of the past five years. It is true that from those reports it would appear the white race has supplied a preponderating number of victims. As the white races alone publish statistics of epidemic visitations, we have little means of knowing what other races may have been involved. An epidemic in the Island of Nauru (which is situated north of Australia and near the equator) attacked 470 natives and 220 imported laborers, of whom a proportion were Chinese. Of the 80 white persons included in a total population of 2330, only 3 were attacked.

Geographic Distribution.—The great majority of epidemics of infantile paralysis of which we have any record occurred in the temperate zone. North Europe, the United States, and Canada were most affected. Of the European countries, Norway and Sweden have had repeated epidemics and of recent years very extensive ones. Germany, Austro-Germany, France, Italy, and England have had more or less severe visitations, and there is little probability that other European countries have escaped, since the disease became pandemic in 1908.

In the Western Hemisphere epidemics have occurred from Toronto to Cuba, and from Boston to the Golden Gate. Florida, in the semitropics, and Cuba, in the tropics, were visited.

The seacoast of Australia has suffered from a number of epidemics, and the Island of Nauru, north of Australia, was swept by an epidemic in January, 1910, which attacked 30 per cent. of the inhabitants. Nauru is in the equatorial belt. It is also geographically isolated, and the infection must have been transported to the island. Is it probable that Australia and Nauru, alone of Oceanica, were infected?

Tubby and Jones, of Liverpool, state that many of the children brought to them with infantile paralysis had been born in India or other tropical countries, and had passed the first three years of their lives abroad, and add that the prevalence of the disease among English children in India is remarkable.

Hill, of Minnesota, relates information from a British East Indian nurse to the effect that poliomyelitis is very common among the native children during the hot weather, especially at the end of the dry season; to which he adds the comment that these children are of a notoriously low nutritional status, and acquire the disease at a season notorious for intense, overwhelming heat, dryness, and dust. I would add to the comment that the bedbug is an ever-present pest in British East India, and the high-caste native will not destroy nor allow destroyed any form of animal and insect life. The East Indian nurse is said to have stated that white children, belonging chiefly to the families of officials, better nourished and usually taken to the hills during the hot season, seem to escape the infection almost entirely. Dr. Hill states that the literature consulted makes no note of this.

A personal communication from Dr. Hernaman-Johnson, of England, states, "I have noted that epidemic palsy prevails in river valleys when enteric is common."

Age Incidence.—There is a wide variation in the reports of investigators regarding the age most liable to be affected, which is shown in the following table:—

Ages.	Wickmann, Sweden, 1905.	Manning, Wisconsin, 1908.	Lovett, Mass., 1909.	New York City, 1907.
0 to 5 years.	350 40.6%	74 49.8%	440 71.5%	660 90.5%
6 and over.	518 59.4%	76 50.2%	175 28.5%	69 9.5%
Total number of cases.	868	150	615	729

A wide discrepancy is noted in the first figures and the last, Sweden reporting 59.4 per cent. of her 868 cases as 6 years and above; while New York City reported only 9.5 per cent. of 729 cases as over 6 years of age. As Wickmann's report was the result of a careful survey of the entire field undertaken by order of the government, it seems probable that it is the most nearly accurate. A large proportion of cases among young adults, and fatal cases of adults, in America, have hitherto remained unreported, because of faulty diagnosis.

The tables of age incidence of this disease in America will be modified as more and more careful records are kept. New York State, in reporting the 227 cases in the State for 1910, already shows a striking difference in age-incidence percentages, 128 of the cases, or 56.4 per cent., being 4 years of age or under, while 99, or 43.6 per cent., were 5 years old or above.

In regard to this point it is of interest to note the report of a physician practising in a small community. The

man at work in a village or hamlet sees every case in that area. I am inclined to regard with special interest such reports from a careful observer.

Dr. H. Prill reported 11 cases from Augusta (population 1400), Wis., 1908:—

2	years of age....	1		
5	" "	1		
6	" "	2	18.1% below 6 years of age.	
8	" "	1		
12	" "	3		
23	" "	1		
25	" "	1		
32	" "	1	81.8% above 6 years of age.	
	Total	11		

Dr. P. Riley reported 13 cases from Elk Mound (population 94), Wis.:—

2	years of age...	2		
5	" "	1	23% below 6 years of age.	
6	" "	2		
7	" "	1		
9	" "	1		
11	" "	3		
17	" "	1		
19	" "	1	76.9% above 6 years.	
23	" "	1		
	Total	13		

(Personal reports to writer.)

These groups are small, but suggest that we labor under some misapprehension in believing this malady to spend its maximum force on small children.

(Sheppard.)

Age.	Cases.	Deaths.	Mortality (per cent.).
From birth to 12 months, inclusive .	117	11	9.40
2 years	61	8	13.11
3 years	98	10	10.20
4 years	69	10	14.49
5 years	51	2	3.90
6-10 years, inclusive	93	7	7.52
11-20 years, inclusive	69	7	10.14
21-30 years, inclusive	28	5	17.80
40 years and over	15	2	13.33
Totals	601	62	..
Average mortality	..	..	10.31

Period of Dentition.—It would seem that being a teething child, *i.e.*, a growing animal in a period of acute development, enormously increases susceptibility to any infection. Poliomyelitis, with its pathologic affinity for the ganglionic motor cells, finds ready victims among children whose developmental period and tremendous activity render these areas correspondingly vascular. Lessened resistance due to the rapid growth can little check the toxic organism, and the child often succumbs during the stage of invasion, before the formation of germicidal antibodies.

Adolescence.—This period, in young males, is apparently one of increased susceptibility. In 150 cases analyzed in the Wisconsin epidemic, 19 were boys from 11 to 20 years of age.

Adults.—No age is exempt. Although the proportion of cases decreases directly in each decade following the first, numerous cases are reported occurring among adults of young, middle, and advanced age.

Sex.—That males are vastly more liable to this disease is shown by the following table of reports of epidemics in which the sex affected is recorded:—

INCIDENCE OF SEX IN SIX EPIDEMICS OF POLIOMYELITIS.

			Males.	Females.	Total.
1894	Caverly[1]	Vermont	53	34	87
1908	Manning[1]	Wisconsin	91	59	150
1909	Lovett	Massachusetts	365	263	628
1909	Anderson[1]	Nebraska	48	38	86
1909	Hill[1]	Minnesota	193	139	332
1910	Dixon	Pennsylvania	437	336	773
	Totals		1187	869	2056
	Percentage		57.7%	42.3%	

Where age and sex have been reported it is manifest that the disproportion of males affected increases directly with the decade of ages. From 1 to 10 years of age the males are slightly in excess, and the proportional increase in the succeeding decades is roughly shown in the following table of Wisconsin cases:—

SEX INCIDENCE BY DECADES.

	Males.	Females.
0 to 9 years inclusive	64	46
10 to 19 " "	16	10
20 to 29 " "	9	
30 to 39 " "	1	2
40 "		1
48 "	1	
	91	59=150

This excess of males has been commented on by many of the writers on this subject, and the attention of the public drawn to the fact by the outbreaks at two notable

[1] Cases reported are only those where sex was stated.

colleges for men, as well as the several thrashing crews whose members were simultaneously attacked. It seems probable that many sudden deaths among adult males since this disease became epidemic in America, due to the bulbar form of this disease, have been diagnosed incorrectly as resulting from ptomaine poisoning, meningitis, lock-jaw, and apoplexy.

OTHER FACTORS WHICH MAY INCREASE SUSCEPTIBILITY OF HOST.

Concomitant conditions more or less constantly present in epidemics were formerly regarded as direct causes of the disease, while their relationship was usually that of increasing the susceptibility of the individual. Trauma, overexertion, and exposure are often given as causes of an attack of poliomyelitis, while their true relationship is that of lessening or lowering resistance, and in the cases of a stumble or fall the accident belongs properly in the list of prodromata of the attack.

Overexertion is frequently given as a factor in the illness, especially of young adult males. A Dunn County, Wisconsin, farmer told of an extreme nervous irritability and sense of impending disaster which kept him in the field at work until overcome with exhaustion. He knew the grain must be got in, and never worked so hard. A farmer's wife ascribed the illness of her 14-year-old daughter to working in the hot sun, carrying stones from a field. Such labor during the onset of an acute disease would directly devitalize and lessen resistance of the organism. In several recorded cases exertion developed an arrested form of the disease, provoking a late paralysis and death.

Exposure to cold and dampness seems in many instances to have been due indirectly to the effort of a

fevered child to get to water. Swimming, unduly prolonged, is often given. The high fever of the preparalytic stage would send a boy directly to the swimming pool,

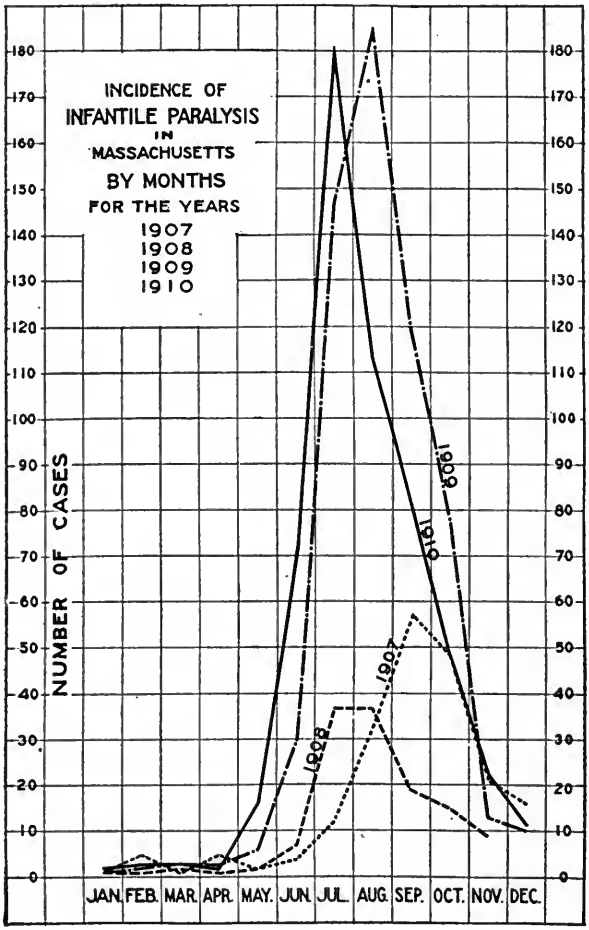


Fig. 15.—Infantile paralysis in Massachusetts. (*Lovett* and *Richardson*.)

especially in those districts where bathtubs are a luxury and total immersion in water hard to come at. A case of so-called exposure was found to be that of a 12-year-old

boy, who at the height of the fever crawled under a garden hose and remained for hours.

A fall is often given as the cause of the attack. Such falls are undoubtedly due to the tremor, inco-ordination, and ataxia that precede, and may be called the aura of the onset. In yearlings and upward to 5 or 6 years of age the co-ordinating centers are still in a state of development and delicate adjustment, which a slight cause will unbalance; the fall is the result and not the cause of the lesion. This ataxia may profoundly affect the strongest adult.

Season.—The epidemics reach their maximum during the summer months of that area in which they are manifest; in the Southern Hemisphere this corresponds to our winter months.

Rainfall.—The deficiency in rainfall during the last eight years has been, of course, very marked, and this factor may be of importance in the spread of the disease. It certainly has to be taken very seriously into consideration if we accept the suggestion of Hill, of Minnesota, that dust, especially dust infected with horse-manure, may be responsible for the transference of this disease. The deficiency of rainfall in Massachusetts has varied since 1904, when it was 1.35 inches, to 9.42 inches in 1910. With the deficiency of rainfall during each year of this period has occurred a sort of cumulative effect, which makes this deficiency relatively more important each year. A study of the deficiency rainfall shows that the largest number of cases have not occurred on the driest years in Massachusetts.

DEFICIENCY RAINFALL (1904-10).

Year.	Cases in State.	Actual (Inches).	Normal (Inches).	Deficiency (Inches).
1904	..	43.81	45.16	—1.35
1905	..	37.60	..	—7.56
1906	..	43.21	..	—1.95
1907	234	44.49	..	—0.67
1908	136	37.61	..	—7.55
1909	923	42.10	..	—3.06
1910	845	35.50	..	—9.42
	..	..	..	—31.56

(Lovett and Richardson.)

FACTORS INCREASING VIRULENCE OF VIRUS.

These factors are unknown. We know that in the experimental production of poliomyelitis in the rhesus ape, the mortality rate, which was low in the first experiments, gradually climbed as a long series of the animals were successively injected from the preceding series, until the rate became 100 per cent. This would indicate that the micro-organism, obtained *post mortem* from a child victim, was steadily enhanced in virulence by culture *intra vitam* in an extremely susceptible host.

Insanitary conditions, by lowering the resistance of the individual, might contribute directly to an increased virulence. Two cases, brothers, both fatal, that assumed an appearance simulating septicemia, were housed in the most insanitary environment seen during the Wisconsin epidemic. The house was built on made ground, in a marshy area. The well and privy were only twenty-seven feet apart, and other hygiene as deplorable as possible.

Time in epidemic, late. Numerous reports of a mortality rate increasing progressively with the continuance of the epidemic have been noted.

CHAPTER IV.

Pathology.

AN AUTOPSY.

THE presence at autopsy table, in the case of an obscure or unknown disease, is more valuable than any instruction or monograph. There may be many physicians who have had no opportunity for conducting or attending such an autopsy. It is so delicate a matter to ask grief-stricken parents to allow that which they consider mutilation of their beloved dead, the physician will make the request only when he is assured, first, that the need for the public weal is most urgent, and, second, that the examination will be conducted with the aid of every modern method of research.

In the recent literature of poliomyelitis, there appeared a graphic report of an autopsy so typical and instructive in all its bearings that its perusal is second only to attending an autopsy conducted by a great pathologist.

The case is typical of many hundreds occurring in America since 1906, not of a member of the poor, obscure, and ill-nourished classes, but from that class of the well-to-do and luxurious whose homes are models of sanitation, and whose children have every possible physical advantage, including the removal from probable areas of contagion during epidemics.

It is to be noted that the case is that of a young male, well developed for his age, who previous to the onset of this disease enjoyed perfect health. The case was reported by Dr. Colin K. Russell, neurologist to the Royal Victorian

Hospital, in the Montreal Medical Journal for July, 1910:—

G. E., a well-developed boy of 10 years, had been spending the summer with his parents among the Laurentian lakes, and previous to the onset of the present illness enjoyed perfect health. Early on the morning of August 5th he complained of headache, and his mother noticed his breathing was more rapid than usual. He spent some time the previous day, which was very hot, swimming, and then lying out on the roof of the cottage in the sun, where there was a little breeze. Just before sunset he and some friends paddled some considerable distance down the lake; after sunset he did not paddle, but lay in the bottom of the boat. He was scantily clad in the light clothes he had worn during the heat of the day, and on being questioned said that his feet were cold. The pain in his head and the back of the neck continued during the day of August 5th. His temperature was 100° F., and he would not take nourishment.

The following day he developed flaccid paralysis of the lower extremities, with loss of reflexes; the arms, later in the day, also became paralyzed, especially the left. Beyond the pain in the head and neck he did not suffer and his mind was clear, respirations still very rapid, but the temperature had fallen to normal and he took some light nourishment. The next day there appeared to be some improvement in the arms, but otherwise no change from this on, except that the respirations became more and more difficult. When he was seen by Dr. Fraser Gurd for the first time on the fifth day of the disease there was paralysis of the intercostals and the diaphragm; life was dependent on the extraordinary muscles of respiration only, and, as was expected, the patient died that night of respiratory failure.

The autopsy was performed at the home of the parents ten hours after death. The brain and cord were removed, the other viscera could only be examined *in situ*, and showed no abnormality. There was nothing to be noted about the calvarium, the dura was of normal color and glistening; there was no congestion either over the cord or brain.

On removal the brain looked large for the size of the head, and it weighed 1550 grams. The superficial vessels were not particularly engorged, but the surface of the brain had a peculiar *slightly*

bluish-gray color; on section the edges evert, showing the capillaries of the *cortex somewhat engorged in places.*

Cord.—The dura was covered posteriorly with a thick layer of fat. The dura itself was healthy and glistening. *On palpation through the dura the cord felt extremely hard, especially over the lumbar and dorsal regions.* On opening the dura the vessels of the pia-arachnoid were slightly congested. The whole cord from the lower end of the cervical enlargement to the sacral segments has the appearance of *having been wound carelessly but tightly with fine thread, showing everywhere little, irregular ridges and bulges, evidently the swollen cord structure bulging through the strands of the pial tissue.* There was some slight cloudiness of the pia-arachnoid over the posterior surface. On section, *the edges evert to a marked degree,* and *the anterior-horn region looks softened, swollen, and almost diffluent; that is, in the lumbar, dorsal,* and to a less degree in the lower cervical region.

Second Sacral Segment.—Microscopically, there is infiltration of the meninges with round cells equally well marked all around the cord. This infiltration does not affect the nerve-roots. The superficial vessels are congested and surrounded by lymphocytic exudation; those of the cord substance are similarly congested and show the same surrounding round-celled exudation. The infiltration of the gray matter is also present, but not to such a marked extent as in the previous case. Many of the *nerve-cells have disappeared,* but a few still remain and retain their normal appearance. The congestion and the *surrounding exudation* are quite general, affecting those vessels coming in from the periphery of the cord as well as the branches of the anterior spinal artery.

Fourth Lumbar Segment.—The same picture here; slight meningitis, marked congestion of the vessels of the substance of the cord, with an occasional small rupture and hemorrhagic extravasation. The same perivascular exudation of lymphocytes and round-celled infiltration of the gray matter. The ganglion cells have entirely disappeared.

Ninth Dorsal Segment.—An identical appearance to those already described. Only a few nerve-cells in Clarke's column on the left side remain.

Eighth Cervical Segment.—The infiltration of the membranes is less marked than in previous sections. It is present to the greatest extent in the anterior fissures and follows the course of the vessel

into the left anterior horn; the vessel is congested, but shows no evidence of thrombosis. The congestion of the vessels in the substance of the cord is general, but the perivascular, round-celled infiltration is by no means so marked as in the previous case, nor is the infiltration of the gray matter nearly so extreme. None of the ganglion cells is recognizable.

Fifth Cervical Segment.—Presents an almost identical picture to that just described, save that there are one or two ganglion cells still recognizable in either horn.

Second Cervical Segment.—Presents a similar appearance, save that the round-celled infiltration is again more marked, especially around the vessels, but also in the gray matter.

Lower Medulla.—The meningeal infiltration is here present, and there is marked generalized perivascular lymphocytic exudation. The nerve-cells, however, appear quite normal.

Right Postcentral Convolution.—Marked congestion of the vessels of the meninges, with surrounding exudation of the lymphocytes; the meninges show a slight degree of infiltration; the cortex appears quite normal.

Right Olfactory Lobe.—Nothing abnormal, although the vessels of the meninges are congested and show the usual lymphocytic exudation. The posterior ganglia were, unfortunately, not examined.

PATHOLOGIC ANATOMY.

Acute Anterior Poliomyelitis results from an infection with a specific micro-organism, which causes a systemic toxemia and localizes in a destructive attack on the cerebrospinal axis, the lymphatic system, and the parenchymatous organs.

Fatal cases of acute poliomyelitis demonstrate an inflammation of not only the gray matter of the cord, but also of the whole cord and pia, the medulla, pons, central ganglia, and the cortex of the brain, always in connection with a like inflammation of the pia mater. It might be expected that in mild cases with symptoms relating only to spinal localization the inflammation would be limited to the cord, but Wickmann, by autopsies on 4 such mild

cases, *"where death occurred during convalescence,"* found evidence of the process throughout the whole nervous system.

The lesions of the central nervous system invariably begin in the delicate and highly vascular pia mater, and ramify throughout its numerous infoldings into the fissures of cord and brain. The process involves progressively the lymph-spaces of the covering of the entire cord, and the invasion continues by way of the lymph-spaces of the vessel sheaths as they penetrate the cord. These vessels, on cross-section, show a girdle of round cells. The numerous round cells crowding the pial lymph-channels and perivascular spaces now proliferate in the tissue spaces of the cord. A few leucocytes are seen during the early stage of this process. They disappear, to be replaced by lymphocytes and proliferating endothelial and glial cells, which, later still, are found in company with extravasated red blood-cells. Capillary congestion, stasis, and hyperosmosis take place, followed by degeneration of the vessel walls and minute hemorrhagic extravasation. Stasis edema and compression result in degeneration of the interstitial tissue, and softening, vacuolization, and final demolition of the ganglionic neuron.

The inflammation in the gray matter of the cord corresponds to the inflammation of the pia mater of the same level. No isolated foci are found in the cord, the lesion in the gray matter being invariably an extension by continuity from affected blood-vessel sheaths which have traversed an infected pia mater. There may, however, be an infiltration of the pia-arachnoid and its vessel sheaths extending perceptibly into the cord, with no invasion of the myelin tissue. This, as Wickmann, the observer, points out, is one of the conclusive proofs that the process has

its initiative in the vascular pia mater, following which "*the inflammation thereupon penetrates the substance of the cord secondarily along the vessels to reach its greatest intensity in the cord substance.*"

The misconceptions which arose in the early study of the pathology of this disease were due to the fact that the autopsy material examined was usually secured from a case of long standing, dead from some intercurrent disorder, and the destruction and disappearance of the anterior-horn motor cells, together with subsequent cicatrization and distortion of the cord, were reasonably supposed to be the primary lesion.

MENINGES OF BRAIN AND CORD.

Dura.—The earliest changes in the cerebrospinal axis take place in the meninges. After invasion of the pia-arachnoid, the virus, traversing its prolongations and infoldings, penetrates the nervous tissue of cord and brain.

The dura mater is usually normal in appearance and color, and not congested. It is usually free from adhesions. In a healthy young male dying in the acute stage, it was found to be normal in appearance, glistening, and the dura of cord imbedded posteriorly in fatty tissue. A dura adherent to vertex is reported at autopsy of a male, 24 years of age, dead on eighth day of illness from respiratory failure.

The dura, much thickened and closely adherent to the pia-arachnoid from the seventh thoracic segment to the first lumbar, completely occluding all communication for fluid from the cord or encephalon above this point, was observed in the autopsy of a girl of 13, dying in the fourth month succeeding a complete paralysis of all skeletal muscles below the waist line.

Pia-arachnoid.—The inner membranes of cord and brain show a pronounced vascular infiltration in all cases dying during the acute stage. It is probable that, in the mildest of ambulant or so-called abortive cases, there is in the stage of onset an edema and lymphocytic infiltration of these delicate structures. The extent of the infiltration is proportionate to the vascularity of the area, and is more intense on the anterior surfaces of the cord where the pia accompanies and incloses the anterior spinal vessels, both arteries and veins, in the anterior fissure. It has been noticed as extreme in the *formatio reticularis.* The meningitic inflammation involves the pia-arachnoid, which extends to and infolds the posterior root fibers and spinal ganglia.

This infiltration is found to be most abundant in the sacral and lumbar regions of the cord, not so evident in the dorsal region, and again more marked in the cervical region. The blood-supply in these portions of the cord is proportionate to the motivation of the corresponding musculature. The enormous muscle mass of hips and legs has its innervation from lumbar and lumbosacral levels. The musculature of torso is much lessened in actual bulk, as well as activity of movement. The centers for innervation of the shoulders, arms, and hands again call for an increased compensatory nutrition. Other pertinent factors would include (*a*) the ceaseless activity of the young child and adolescent male, and (*b*) the period of growth of musculature.

These two last-mentioned conditions would combine to increase the determination of blood to, and perhaps lessen resistance of, the motivating cell for which the virus of poliomyelitis appears to have a pathologic affinity.

When, on sectioning the meninges, the swollen and edematous cord is exposed, transverse fibers of pial tissue

are seen to divide the surface of the cord into many irregular crosswise ridges, as if the cord from the cervical enlargement to the sacral segments had been snugly wound with fine thread.

The same evidences of meningitis serosa are found in the pia-arachnoid as it extends over, and penetrates the infoldings of, the medulla, pons, and cerebellar and cerebral cortex. This inflammatory condition decreases in degree as the vertex is approached. It is evidenced macroscopically by an intense hyperemia of the vessels and tissues of the pia covering the base, and the central lobe of the little brain, and entering the Sylvian fissure. In cases exhibiting bulbar symptoms, an edema of the base of the fourth ventricle accompanies this hyperemic congestion.

CEREBROSPINAL FLUID.

The virus of poliomyelitis has been demonstrated to be present in the cerebrospinal fluid of monkeys during the preparalytic stage. As its presence has not been determined in the spinal fluid of man or monkey after the paralysis has occurred, it is supposed to have passed into the cord. It is possible that under certain conditions its life cycle is self-limited.

With the onset of the disease the spinal fluid is obtained under slight plus pressure, indicating an increase in the quantity of the fluid. With a violent onset with marked meningism the pressure may increase to an extreme degree, and the fluid will spurt with considerable force when the spinal canal is tapped. The normal pressure flow is drop by drop, with the drops moderately slow in forming.

The spinal fluid is clear at onset, becoming opalescent in the preparalytic stage, and entirely clear as paralysis

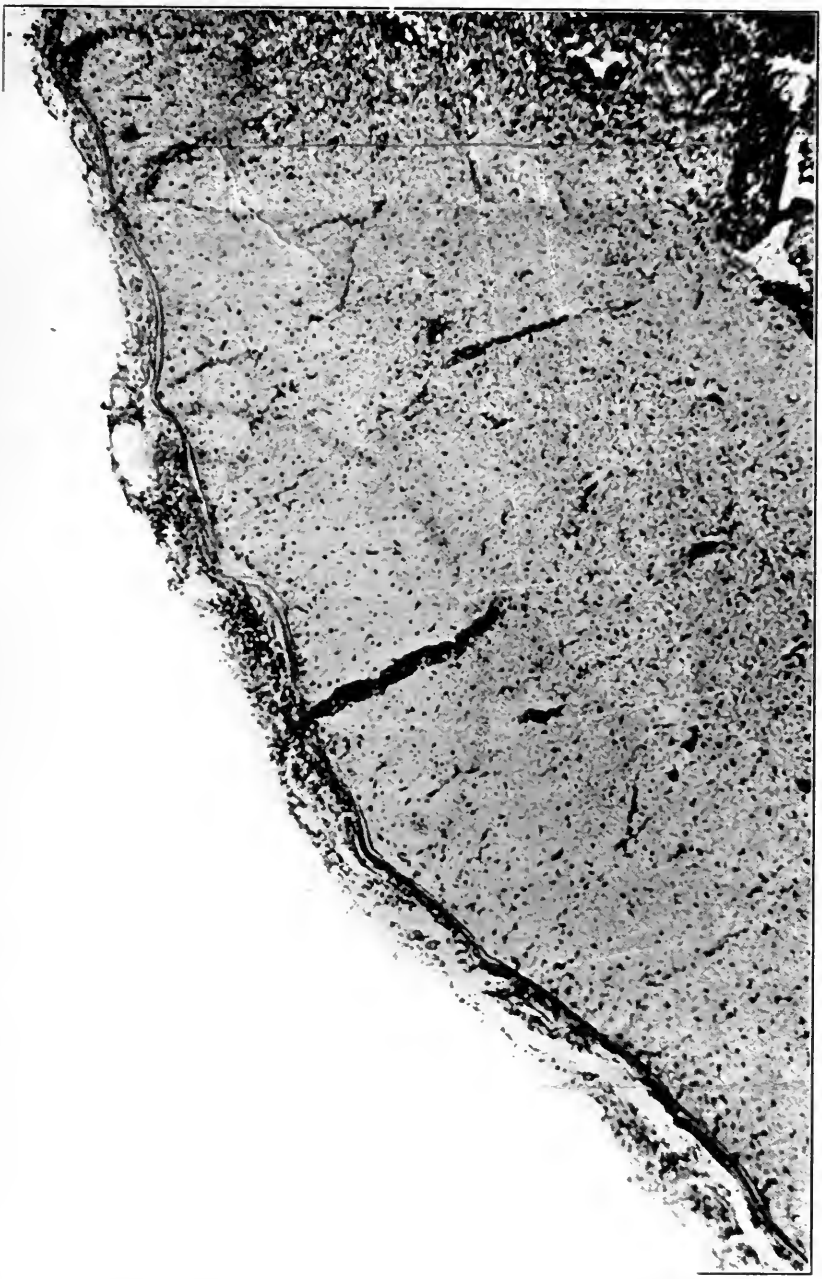


Fig. 16.—Acute poliomyelitis of spinal cord (human). Meningeal and perivascular infiltration. (*Skoog,* Bulletin of Kansas State Board of Health.)

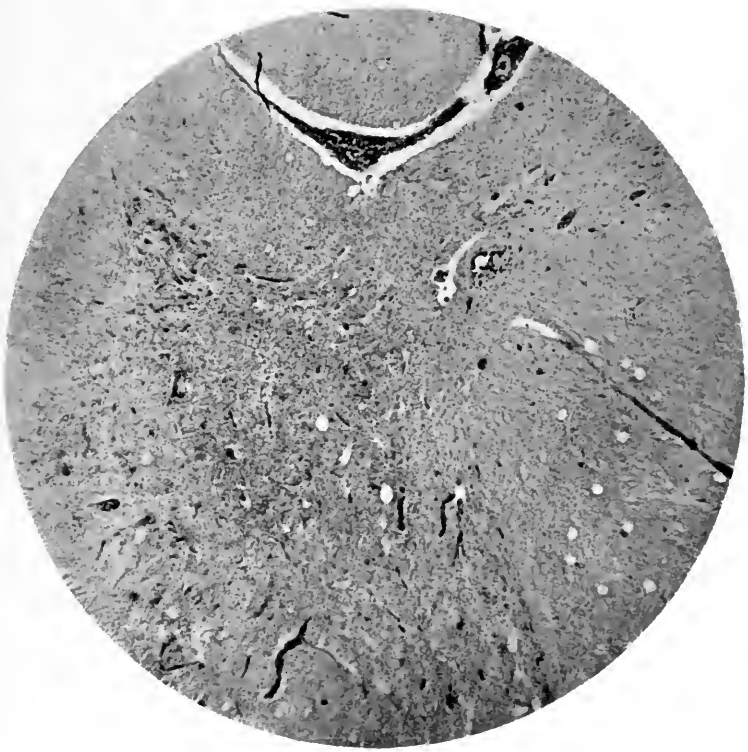


Fig. 17.—Inflammatory process extending throughout anterior horn.

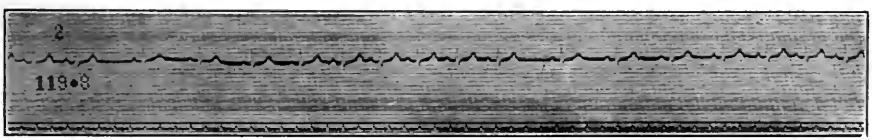


Fig. 18.—Electrocardiogram of arrhythmia regularly seen in fatal cases. (*Peabody, Draper,* and *Dochez.*)

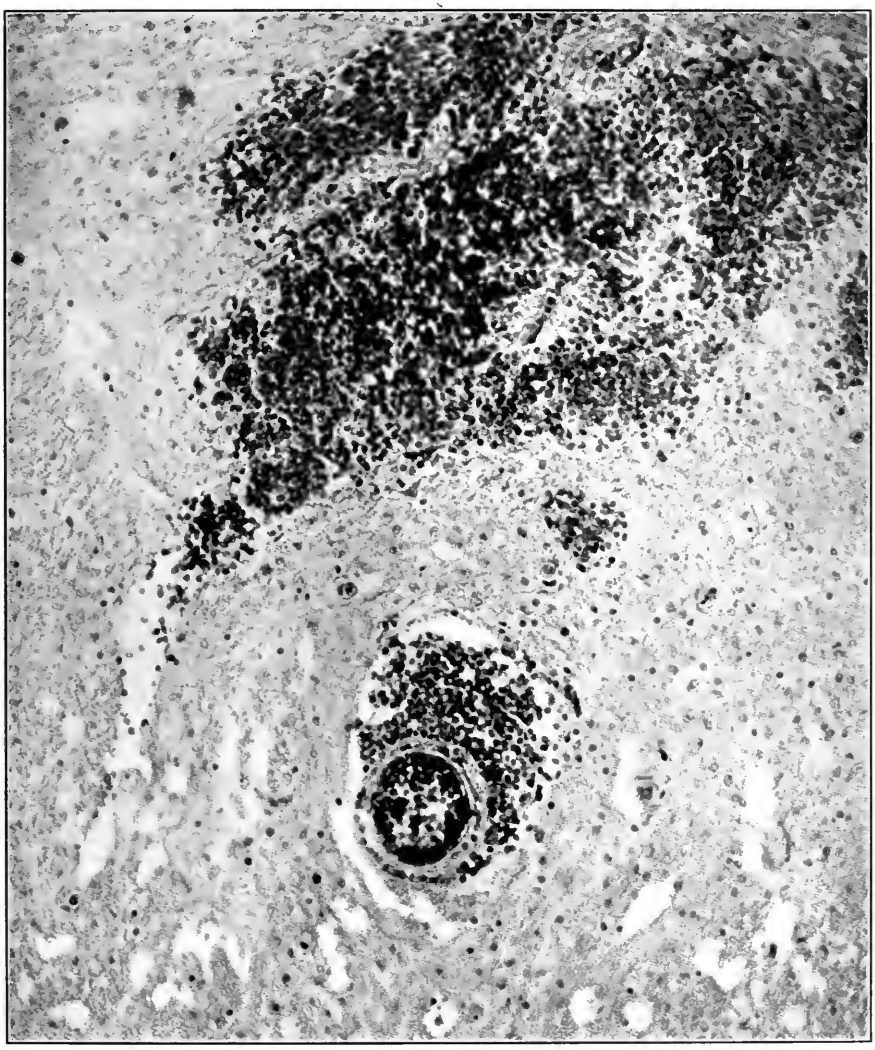


Fig. 19.—Diffuse areas of hemorrhage in the gray matter of the cord. (After *Peabody, Draper,* and *Dochez.*)

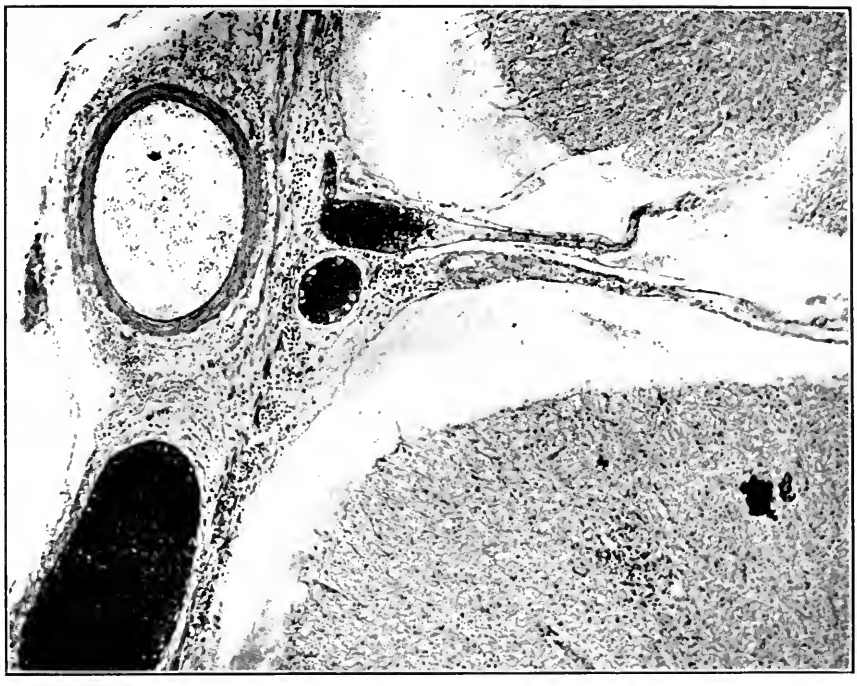


Fig. 20.—First sacral segment, showing infiltration of mononuclear cells in the pia mater at anterior fissure and in the sheath of vessels.

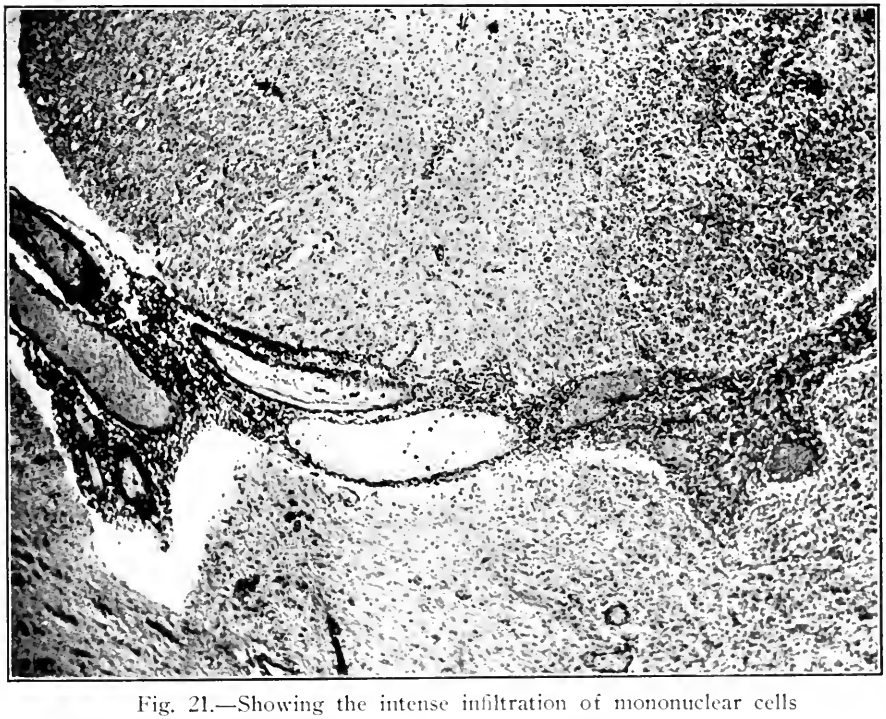


Fig. 21.—Showing the intense infiltration of mononuclear cells around the anterior spinal vessels, both in its walls and in the adjacent gray matter of the anterior horn. This vessel is a continuation of that shown in Fig. 20.

(From Report of the New York Committee.)

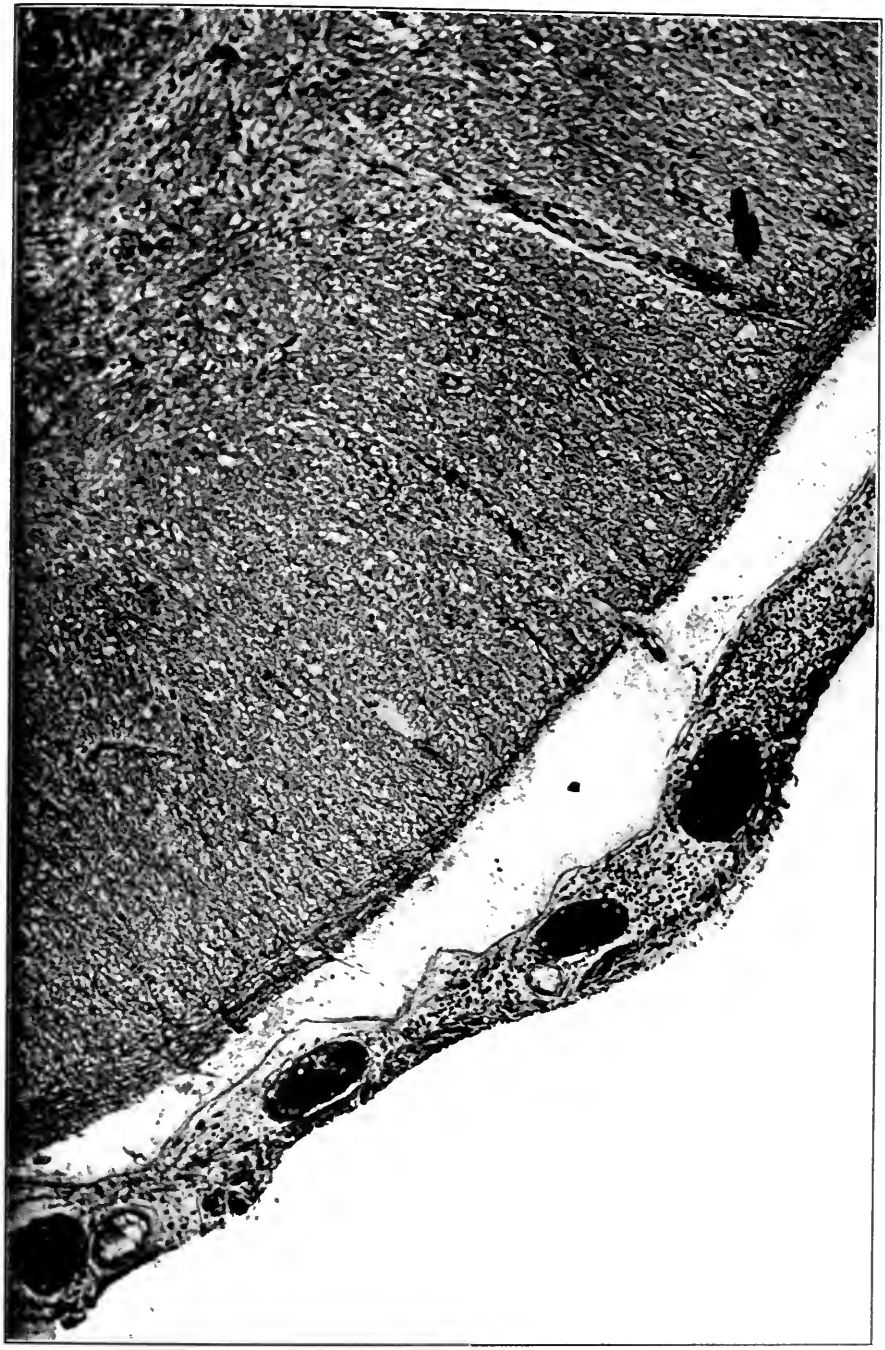


Fig. 22.—First sacral segment, showing the intense infiltration of mononuclear cells in the pia mater. (Epidemic of 1907.) (From Report of the New York Committee.)

ensues. This preparalytic opalescence is a diagnostic and prognostic point, confirmed by Frizzell.

The slight milkiness is due to an early phagocytosis, of which a few white cells, at first polymorphonuclears and then lymphocytes, are manifested in the fluid.

There is an increase in the proteid content of the fluid, and the fluid may coagulate spontaneously, due to an increase in the fibrin.

THE CORD.

Palpation of the cord, before the dura is incised, discloses its extremely hyperemic and edematous condition. Resistance to pressure is increased, and in rapidly fatal cases the cervical and lumbar portions of the palpated cord may feel hard as a lead-pencil.

On incising the dura the cord will be seen to present many delicate constricting bands of pial tissue, between which the swollen and edematous myelin structure of the cord is elevated in transverse ridges.

On section of the cord the cut edges evert, and an unusual surface moisture is apparent, due to edema. Small hemorrhagic areas are seen by the naked eye, and the motor columns may show evidences of softening.

Microscopically, the early changes in the cord consist of a hyperemia, intra- and extra- vesicular; a perivascular infiltration similar to and continuous with that of the pia mater; a round-celled infiltration seen as a collar about the cut vessel walls; here and there foci of cells heaped together, and a general diffuse infiltration throughout the myelin tissue, gray and white. Hemorrhagic areas occur more frequently in the gray nervous tissue. The gray matter in general is more affected than the white, although no part of the cord at cervical and lumbar levels escapes scot free. In direct proportion to the vascular supply the

dilatation and engorgement of the vessels and attendant infiltration of myelin are more marked in the anterior horns than the posterior, and, at the high levels of function, the cervical and lumbar areas than elsewhere. The anterior spinal veins are as much congested as the anterior spinal

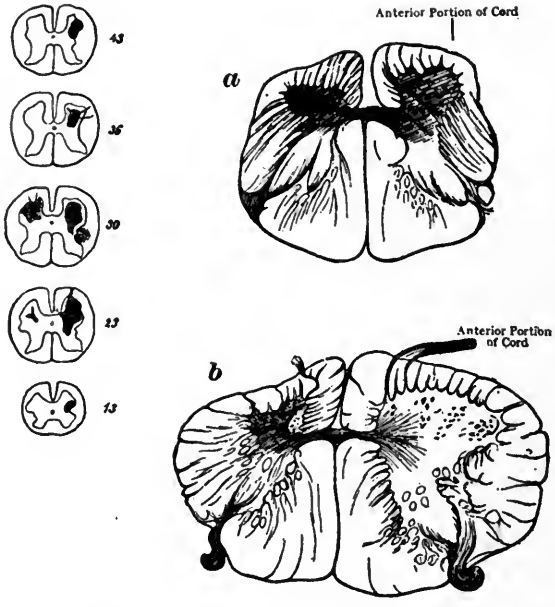


Fig. 23.—Spinal cord with acute spinal paralysis forty-three years after beginning of disease. *a*, section through lumbar (back) enlargement; *b*, section through cervical (neck) enlargement. (After *Charcot* and *Joffroy*.) 13, 23, 30, 36, and 43, localization of diseased area in anterior horns of lumbar enlargement of cord in child 2 years old, eleven months after beginning of disease. (After *Roth*.)

arteries, and some observers have found them to be more involved.

Hemorrhages are due to the action of the virus on the vessel walls.

The secondary effect of this inflammatory process is the damage or destruction of the motor ganglion cell of

the gray matter. The vascular infiltration and pressure edema may temporarily inhibit the functioning of the cell, with a resulting regressive paralysis. When more extensive damage occurs to the ganglion cell it may degenerate and disappear, its place being taken by young glia

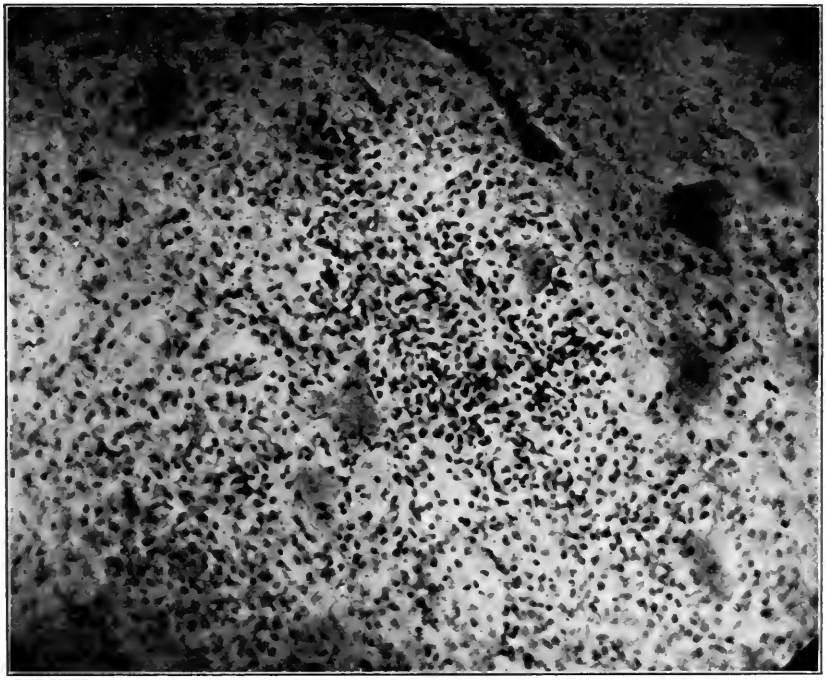


Fig. 24.—Blood extravasation into the motor area of the spinal cord.

cells. No ganglion cell is immune, although the cells of Clarke's column are relatively immunized by their distance from the seat of war, and often escape involvement. The ganglion cell may suffer marked impairment in function before any morphologic change can be determined. Restitution of necrosed ganglion cells does not take place, but the less affected cells regenerate and regain their original function.

The changes taking place in a degenerating ganglion cell include cellular invasion, displacement of the nucleus, vacuolization, disintegration of the nucleus, tigrolysis, destruction of the protoplasmic processes, and necrosis. The axon is the last part of the cell to yield to the destruc-

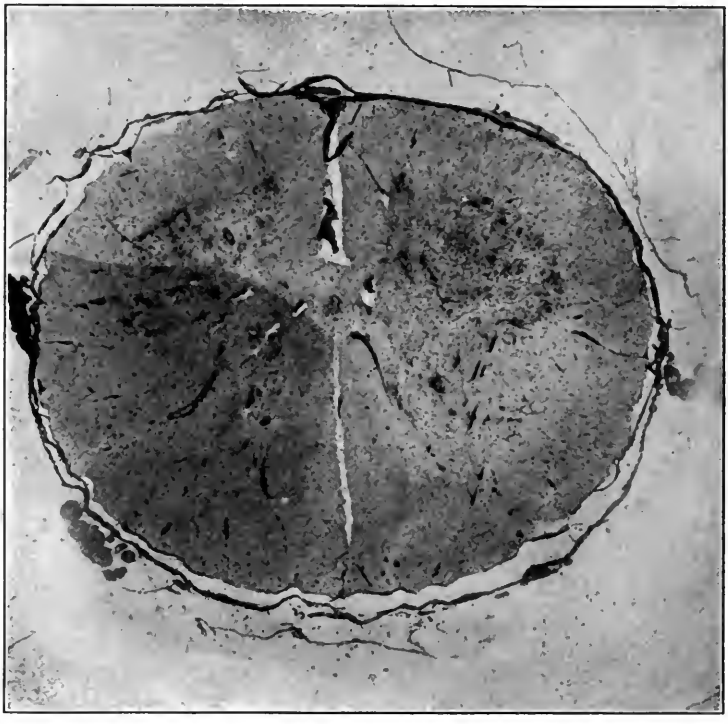


Fig. 25.—General view of human cord (cervical level) in poliomyelitis.

tive process, which accords with the observed fact that no degeneration of the distal nerve-fibers has been determined. Destruction of the ganglion cells always takes place in an area suffering from a furious invasion; the destruction may proceed rapidly over large areas, but in each area there may remain apparently normal cells.

The invading cells, or neuronophages, which gather

in the vicinity to participate in the destruction of the doomed cells, are lymphocytes and polynuclear cells of probably glial origin. The absence of granules from the protoplasm of the polynuclear cell demonstrates their origin to be other than leucocytic. The glial parentage of

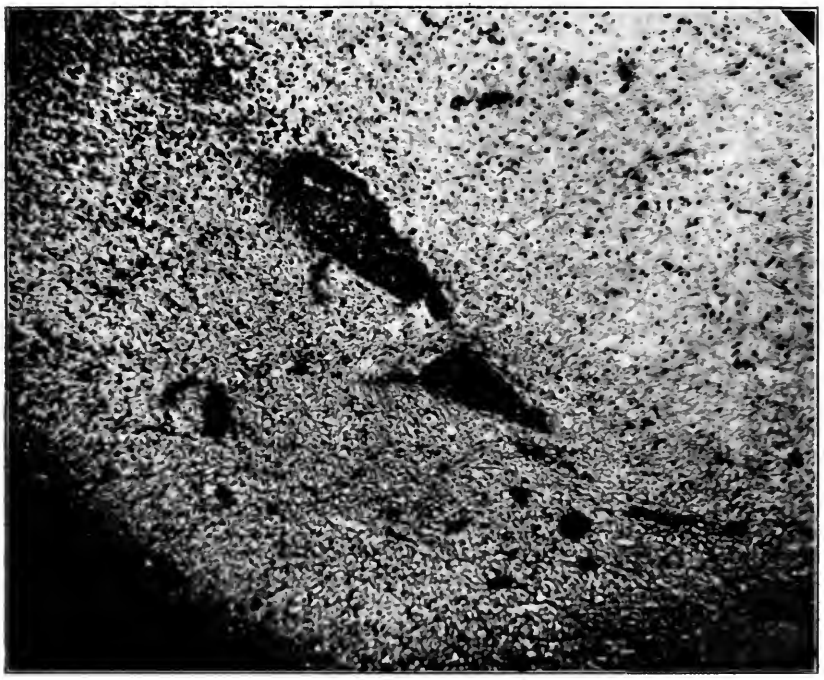


Fig. 26.—Perivascular small round-cell infiltration.

these cells is made probable by the later pathology of the cord in old cases; the cord is seen to be distorted, the anterior horn or horns replaced by a deformitive glial tissue, the connective tissue of the nervous system.

The areas most affected in the cord are the lumbosacral and cervical enlargements. In the cervical region the damage is most marked in the anterior horns, although Clarke's column may be involved. In the lumbar enlarge-

ment the destruction of the posterior gray columns may be as extensive as that of the cells of the anterior columns.

An invasion of the white matter of the cord appears to be a tertiary involvement; some infiltration of the vessel walls and an edema are necessarily constant attendants of

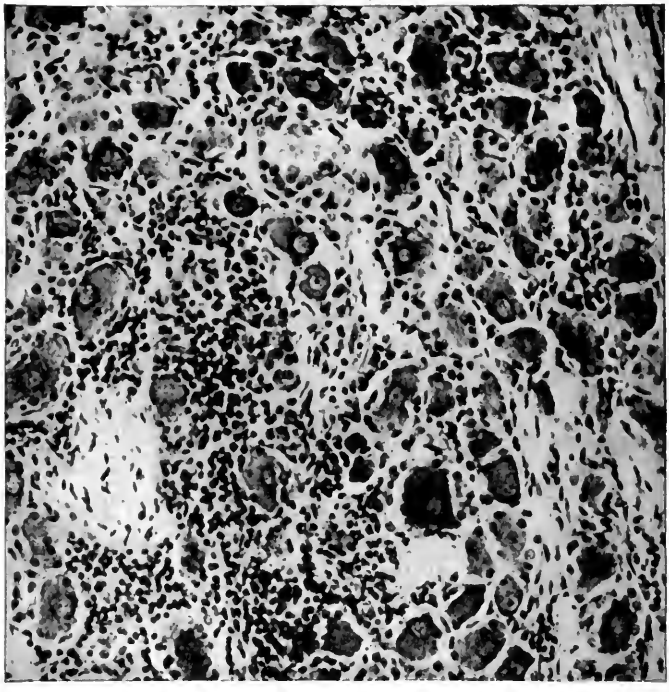


Fig. 27.—Round-celled infiltration and hemorrhagic areas.

the ferocious attack on the adjacent gray columns; if, however, round-celled infiltration and hemorrhagic areas are markedly present in the white substance of the cord, there will have been a corresponding clinical history of incoordination, ataxia, spasticity, and continued exaggeration of reflexes.

Anterior Root Fibers.—Early degeneration of the nerve-fibers from the anterior roots has been observed by

Robertson, of Minnesota. Involvement of the trophic branches would explain the numerous cases of herpes zoster among elderly, undernourished women.

Posterior Root Fibers and Spinal Ganglia.—The membrane covering the posterior root fibers, on their emergence and continuing over the spinal ganglia, shows vascular congestion and infiltration continuous with the meninges of the cord. Spinal ganglia diffusely infiltrated with round cells and edematous are found. More advanced lesions show necrosis of the nerve-cell and disintegration by neurophages. This involvement of sensory root ganglia explains the intense pain of the preparalytic stage.

Peripheral Nerves.—The pathology of the peripheral nerves and their termini has not yet been worked out. Cases simulating acute multiple neuritis are supposed to be due to lesions in the cord. This is yet unproven.

Medulla and Pons.—The pathology of the brain-stem gathers tremendous significance from the fact that a majority of the deaths (90 per cent.) in this disease are due to bulbar paralysis. Involvement of the vital centers in the medulla leads swiftly to paralysis of respiration or cardiac failure.

An intense hyperemia and cell infiltration are found throughout the medulla and pons, extending upward from the cord. This lessens in degree at the higher levels, but is never absent from gray matter of the medulla in all cases that have come to autopsy. The hyperemia, which is constantly contiguous to congested vessels of the pia, may be hemorrhagic. An edematous condition of the tissues making up the floor of the fourth ventricle is found in cases presenting serious medullary symptoms.

The motor cells of the cord have their analogue in the cells of the motor nuclei of the cranial nerves. Infiltration around these cells induces the various regressive cranial-

nerve paralyses. In rare cases nuclear centers are more or less completely destroyed; usually the ganglion cells are damaged or destroyed in small and separate areas only.

The autonomous centers in the floor of the fourth ventricle whose exact *locale* is yet uncertain, which play so large a part in the mechanism and governing of the body, are all subject to the most extreme disorder from this invasion.

The vagus center registers its first irritation with tachycardia, or arrhythmia, or both. The tachycardia is usually extreme. There are undoubted cases where the paralysis of the heart precedes the respiratory paralysis.

Celestine M., male, 13; paralysis of flexors and extensors of both legs. Died sixty hours after onset of heart-failure. Medulla involved. Respiration unaffected. (Personal report from Dr. Boothby, Hammond, Wisconsin.)

The excessive sweating, which attends the fever in some cases, implies an involvement of the vasomotor centers. The uncontrollable projectile vomiting is also induced by irritation at the centers. The heat centers may be profoundly affected.

Elbe F., 18 months. Acute onset May 26, 1908. Partial paralysis of both arms and both legs. Left facial paralysis. Convergent strabismus of left eye. Unable to hold head erect. Motion returned to arms on twelfth and limbs on fourteenth day. Eighteenth day, hypoglossal involvement. Twenty-fourth day, twitching of arms and legs. Twenty-sixth day, spastic contractions of hands. Thirty-eighth day, temperature rose to 108.2° F., rectal. Death on fortieth day, rectal temperature one hour previous being 110° F., pulse 154. Diagnosis, infantile paralysis of cerebral type. (Personal communication. Dr. G. H. Fellman, Milwaukee.)

Vital centers may be suddenly and overwhelmingly attacked. In fatal cases a few hours only may elapse between onset and death.

H. P. K., 7½ months. In same house with cousin, T. P.; paralyzed in May. June 1st, onset. Fever, convulsions, stupor, eleven hours from onset to death. (Personal communication. Dr. Henri B. Cole, Blk. River Falls, Wisconsin.)

THE BRAIN.

Diminishing in intensity from the bulb forward and upward, infiltration can be traced over the cortex of the brain to the vertex. Hyperemia, infiltration, foci of round cells, and edema have all been observed in the cortex, and to a lesser degree in the white substance of cerebrum and cerebellum. The prognosis as to life in the encephalic form is much more favorable, and these cases have not provided much autopsy material. In cases presenting a clinical picture of an encephalitis the process would be marked in the pia-arachnoid and the subjacent cortex. Spastic monoplegias, convulsions, epilepsy, hydrocephalus, and mental deficiency, with acute tremor, acute ataxia, and nystagmus if the cerebellum is involved, will be found associated.

THE CEREBELLUM.

Involvement and infiltration of the cerebellar substance at the decussation produce a tremor due, from irritation, to the alternate action of groups of muscles and their antagonists. This tremor is slow, coarse, and regular, involuntary, but of the intention type, and associated with a spastic and relaxed musculature. Its exact analogue has been long recognized as paralysis agitans.

In 1899 the late Professor Brower presented to his class in nervous diseases an advanced case of paralysis agitans as a typical case, a woman of 60, who gave her own history. She had been acutely ill at 16 years of age with "brain fever." Was subsequently as we saw her, with spastic and atrophic muscles, tremor, and the propulsive gait, which she described in saying that she ascended and descended stairs successfully only by "running." This woman

stated that a brother, acutely ill at the same period, was left a hopeless paralytic. Professor Brower made no attempt to co-ordinate these two cases, beyond calling the attention of the class to the fact that both cases began acutely.

The tremor may extend to the head, tongue, muscles of trunk and extremities. The child may appear to be shivering with cold. In a case of a girl of 16 the tremor was confined to face and tongue; a paralysis of the sterno-cleidomastoid of one side allowed the trembling head to be drawn well toward the opposite shoulder.

An invasion of the cerebellar cortex may be followed by a "wild ataxia." This comes on acutely with associated spastic conditions, nystagmus and scanning speech. It may be veiled by stupor during the acute stage, and become manifest only when convalescence is established and the patient active.

Lesions of the optic thalamus produce an athetoid condition with slow weaving of the affected extremities, and a more or less constant vermicular motion of the torso in children still in arms. Other clinical symptoms of cerebral involvement are usually present, with a cerebral hemiplegia or paraplegia. The following case was seen at the Hospital for Deformities and Joint Diseases, New York City:—

F. B., Yonkers. Well-developed male, 19 months. Walked and talked at 14 months. Acute onset April, 1911. Unconscious nine days. High fever; opisthotonus; strabismus. Five weeks later, paralysis of extensors of both legs; spastic condition of right and left great toes; spastic and relaxed condition of both arms, which are constantly employed in slow, vermicular motion, the hands weaving circles which extend to limit of range above head and posteriorly. Constant, slow, wriggling motion of torso. These movements cease in sleep. Makes no effort to sit, stand, or speak.

Head hydrocephalic, circumference 18¼ inches. Fontanels unclosed. Mentality distinctly disturbed, but recognizes parents. Marked irritability.

CEREBRUM AND CENTRAL GANGLIA.

Infiltration from the involved pial vessels has been traced up and over the hemispheres, including the central gyri. Harbitz and Scheel found inflammatory changes in certain parts of the substance of the hemispheres, as well as the cerebral cortex, and consider that it is present in these localities in all serious and fatal cases. They found anatomical evidence of a very severe degree of cerebral invasion and softening, in the case of a man of 39 years which came to autopsy. The case occurred in the same locality and simultaneously with cases of poliomyelitis, and for this reason, and because the inflammatory process in nature and extent corresponds with that of acute poliomyelitis, it can be classed with certainty with that disease.

Male, 39, presented symptoms of meningoencephalitis, namely, fever, headache, stiff neck, vomiting, convulsive seizures, clouded consciousness, and some rigidity of limbs. Finally coma and death occurred after twelve days' illness. There were no paralyses or pareses. At necropsy, aside from diffuse hyperemia of the central nervous system, there were softened encephalitic foci in the right temporal lobe, and in the gyrus fornicatus of both sides. In addition, the inflammation extended, though with lessened intensity, to the basal ganglia, along the aqueduct of Sylvius, through the entire medulla oblongata, and was even demonstrable in the cord. The inflammation had the same characteristics as that of poliomyelitis; also in this particular, that there were cellular infiltrations in the pia mater. (Harbitz and Scheel.)

Encephalitis of the cerebral cortex is now known and described as polioencephalitis superior. Three classes of cases have been recognized, according to localization of the lesion, as:—

1. Rolandic. (Cerebral hemiplegia.)
2. Frontal. (With mental and moral deterioration.)
3. Occipital. (Blindness, with normal eye-grounds and active pupils.)

DIGESTIVE SYSTEM.

An invasion and inflammation of the tonsils and salivary glands were discovered early in the experimental work with monkeys, and pointed the way to the findings in the lymphatic system as a whole. Strauss, of Cornell, found acute enlargement of the solitary follicles and Peyer's patches of the small intestine, and an acute inflammation of the mesenteric glands. The involvement of these lymphatics was found in every case where the intestines were examined, and in 1 case there was ulceration of the follicles and patches. Inflammation was found present early in serious cases. The acute swelling of the follicles and glands was very marked in a case that died three hours after onset of paralysis.

THE SPLEEN.

The spleen is enlarged and congested, showing a marked hyperplasia of the Malpighian bodies, and proliferation of the endothelial cells.

THE LUNGS.

The lungs are congested, and areas of bronchopneumonia are frequent. Strauss considers this an aspiration pneumonia. Considering the frequency of involvement of the muscles of respiration, with deficient oxidation, and the invasion of the adjacent blood-vessels of the cord, the possibility of a toxic pneumonia must be considered, until the knowledge of the lesions of this protean disease is further advanced.

THE LIVER.

Mild parenchymatous changes have been noted in the liver. Necrosis and disintegration of the liver-cells are reported by Peabody and Draper.

THE KIDNEYS.

The kidneys show congestion, edema, and a beginning degeneration in mild cases, with an acute exudative nephritis in severe and rapidly fatal cases. Clinically, cases supposed to present a retention have, on catheterization, been found to be suffering from anuria. Anuria might result from the profound action of the virus overwhelming the secretory cells of the uriniferous tubules, or from a total inhibition of the centers for innervation of these cells.

THE BLOOD.

Evidence of the destructive action of the living virus throughout the system indicates that it is carried by the blood-stream to all organs of the body simultaneously with its localization in the great central ganglion.

THE VISCERAL LESIONS OF HUMAN POLIOMYELITIS.

During the past summer the organs from 11 children, ranging in age from 3½ months to 9½ years, became available for study. Of the 11 children, 10 died on the third to the eleventh day of illness, and 1 child, having survived the acute stage of poliomyelitis, succumbed two months later to laryngeal diphtheria.

All the fatal cases examined by us showed some and usually a high degree of hypertrophy of the lymphoid tissues. The affection of these tissues was widespread, if not universal, and included the tonsils, small intestine, thymus, and the superficial and deep lymphatic glands. The glands about the trachea and bronchi and those of the abdominal cavity were usually much enlarged and definitely softened in consistence. The mucosa of the large intestine escaped affection, although the mesocolic glands did not. While the cervical, axillary and inguinal nodes do not become prominent, they all showed definite enlargement to the naked eye. The spleen was somewhat enlarged and the appearance was universally altered. The Malpighian bodies were prominent, and the pulp was increased and of dark or grayish hue. Defects in the intestinal mucosa were never observed. The necropsies were performed a few hours after

death, so that the organs were secured in a state free from post-mortem changes.

The characteristic appearances observed in the parenchymatous organs relate to the lungs and the liver. The lungs, in several instances, show within the capillary and other small vessels a considerable number of myeloid cells of the megakaryocytic type. Similar cells are found in some sections of the spleen; but the lesions in the liver are conspicuous and, apparently, constant. They consist of hyaline focal necrosis of liver-cells, followed by regeneration and invasion by lymphoid cells and polynuclear leucocytes. The number of areas of necrosis is remarkably large; the extent of the necrosis varies from a few cells to an eighth of a lobule. The liver-cells appear hyaline, stain deeply in eosin, are coalesced, and are sometimes in process of disintegration. The location of the necroses is remarkable; they are not infrequently adjacent to the portal spaces, but they are far more common about the central and sublobular veins. The smaller ones are readily overlooked; the larger ones resemble the "lymphoid nodules," so called, of the liver in typhoid fever. What is remarkable is the large number present in the sections—a dozen or a score or more of separate areas may occur in an ordinary section. The rapidity with which regeneration is attempted is a striking feature; proliferated nuclei of liver-cells, arranged often in double rows so as to simulate bile-ducts, occur in all the specimens. When it is recalled that the *obvious illness* in some instances was only of *three or four* days' duration, and in most cases was less than seven days, both the *wide extent of the necrotic injury* and of the marked effort at repair are worthy of being noted.

A study in children of the general visceral lesions of epidemic poliomyelitis leads us to place the disease among the affections in which the organs generally suffer injury. The main injury appears to be inflicted on the nervous organs, next on the lymphatic organs, and last on the parenchymatous organs. Of the last, the focal necrotic lesions of the liver are especially impressive. Whether the organic lesions, exclusive of those of the nervous system, are to be attributed to parasitic action or to the action of toxic elements of parasitic origin, is, at present, a matter of conjecture. But the polymorphonucleocytosis of epidemic poliomyelitis is caused not only by the lesions of the nervous system, but also by lesions of the

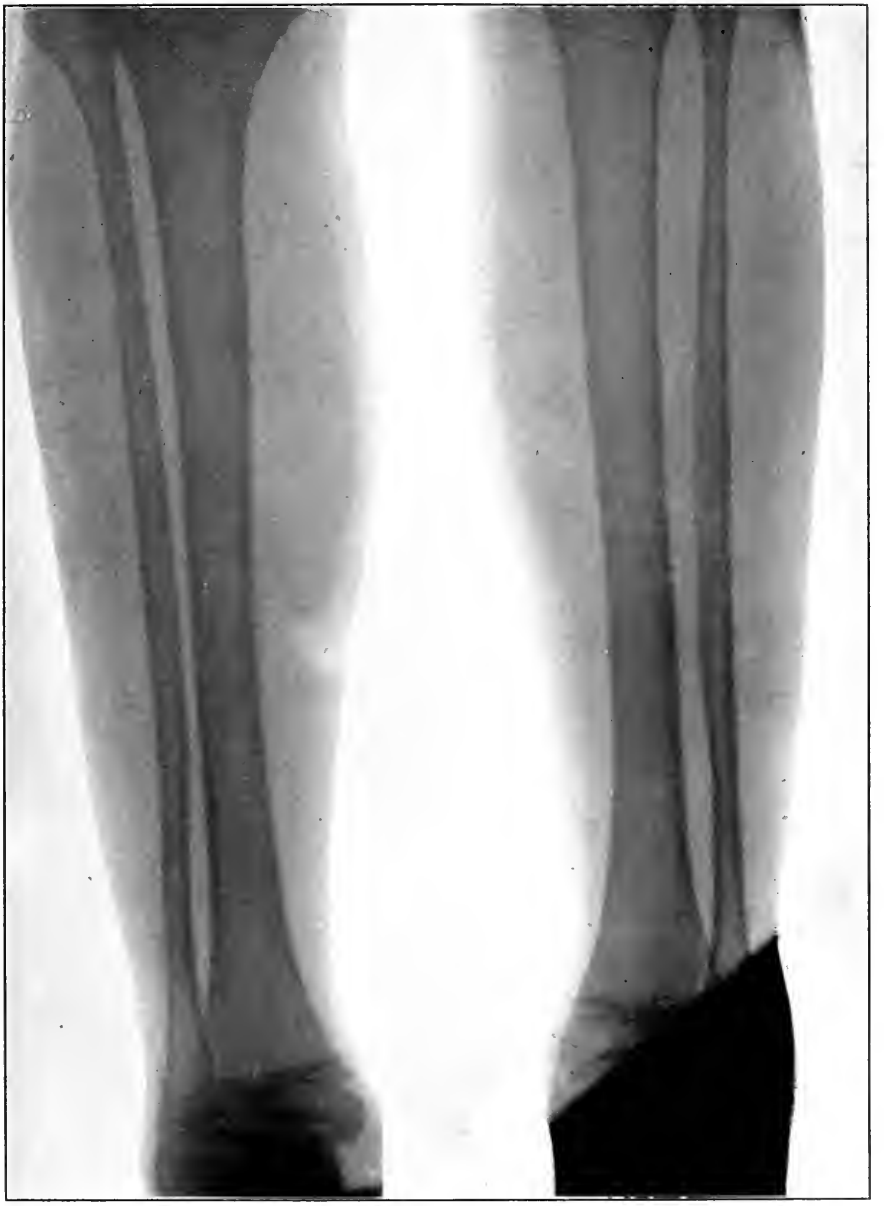


Fig. 28.—Actual shortening of left leg. Note epiphysial line.

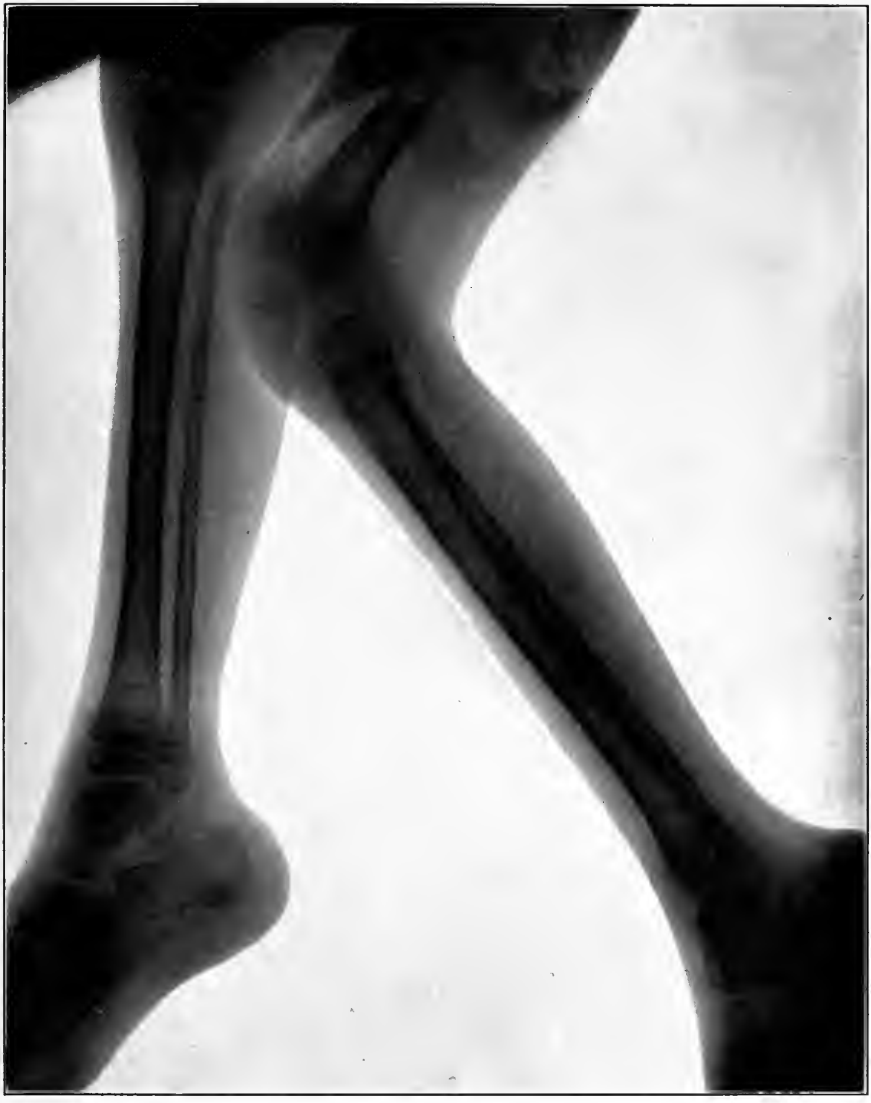


Fig. 29.—Drop-foot and shortening. Compare with X-ray of club-foot (Fig. 31).

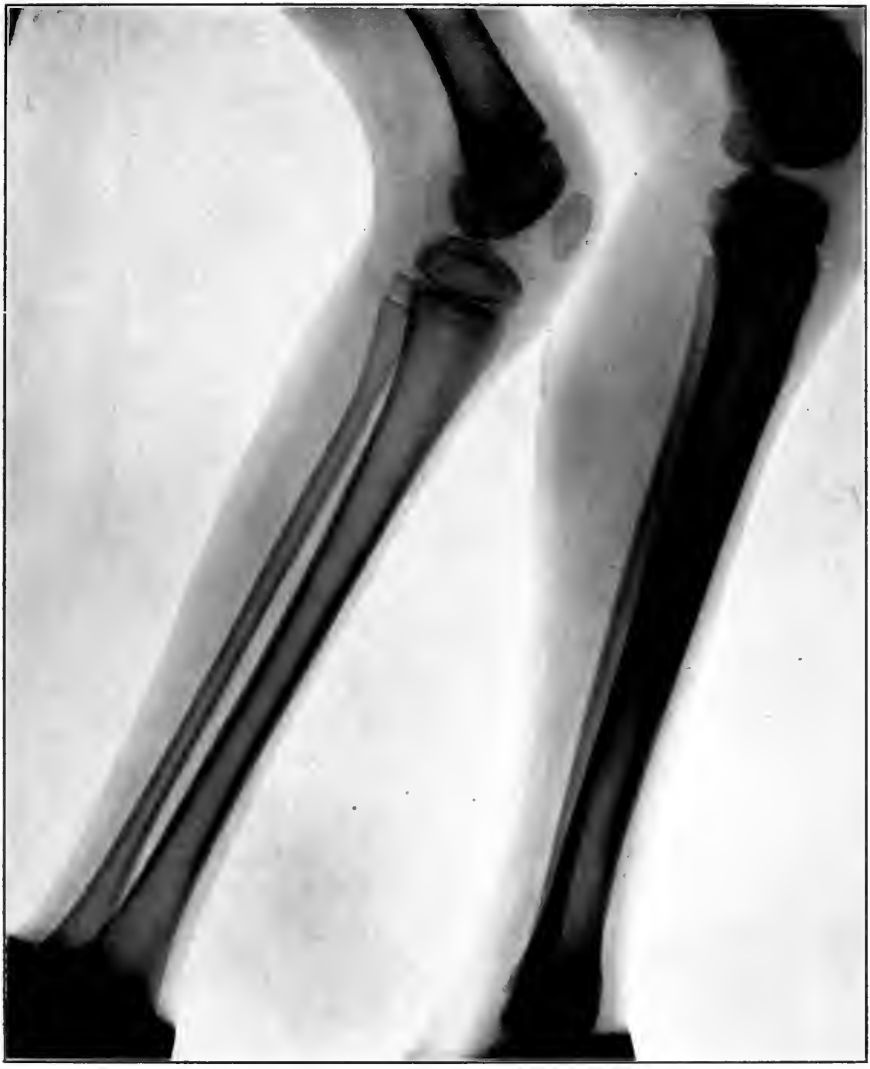


Fig. 30.—Paralysis of right leg, with shortening, at 9, from infantile paralysis at 4 years of age. Note slender, rarefied tibia, and atrophied muscles of paralytic leg.

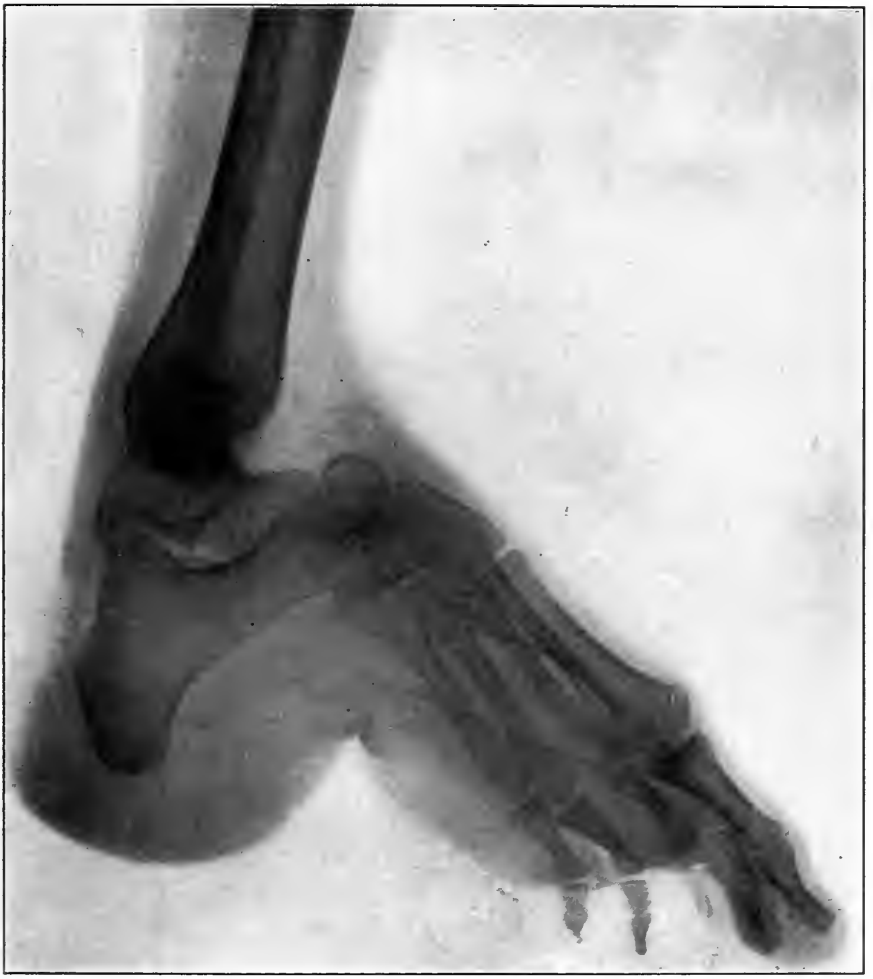


Fig. 31.—X-ray of foot, showing bony deformity.

lymphatic tissues and liver. This consideration will serve to explain certain discrepancies in the cell findings in the cerebrospinal fluid removed by lumbar puncture, and in the circulating blood. (Flexner, Peabody, and Draper, Journal of the American Medical Association, Jan. 13, 1912.)

PATHOLOGIC ANATOMY OF THE CHRONIC STAGE.

The Spinal Cord.—"In the residual stage are found patches of scar tissue, 1 to 2 centimeters in length, with atrophy of the anterior horns or even of half the cord. The nervous material will be found almost entirely absent. The anterior roots at the level of the cicatricial patch are decreased in size." (Vulpius.)

The Muscles.—"Muscles of the affected parts of the limb show marked change. They decrease rapidly in size, and exhibit degenerative atrophy of variable distribution and severity. The normal fibers are distinguished by their bright-red color, while the degenerate ones are of a yellowish-white hue. Separate fibers, of which the bundle is composed, can hardly be distinguished. Areas of streaky degeneration are sometimes seen lying side by side with healthy fibers in a muscular bundle. These are the so-called 'tabbycat' muscles. A considerable deposit of fat may take place in and around the degenerate muscle.

"Certain muscles, though subnormal in size, show a more or less exaggerated red color; these are fibers which have undergone disuse atrophy or overstretching. A faulty position of a limb results in the shortening and wasting of some muscles, while others are overstretched. Compensatory hypertrophy of muscle may take place." (Vulpius.)

The Tendons.—"The tendons also participate in the process of atrophy. This is to be attributed to the disuse rather than degenerative change. They become smaller

and somewhat weaker, and where they are intersected by degenerated muscle they are particularly likely to yield, *e.g.*, in the quadriceps tendon." (Vulpius.)

The Skeleton.—"The changes in the skeleton are very remarkable. The long bones of a paralytic limb are more slender than usual; the outer, compact layer is rarified, and the medulla is reduced in amount. The difference in

Fig. 32.—Club-foot from infantile paralysis.

length is particularly marked, and may amount to several centimeters; a difference of as much as 20 centimeters has been recorded. The bones may acquire a permanent deformity as the result of long-continued faulty position of the joints and the unequal stress that is laid upon them in consequence." (Vulpius.)

The Joints.—"As a rule, the joints are relaxed; the capsule and ligaments are overstretched, the articulation becomes lost, and subluxation follows, or even complete

dislocation, with the familiar changes in the articular surfaces. In other cases one finds more or less marked contracture due to partial paralysis of the muscles; with this is associated unilateral shortening of the capsule." (Vulpius.)

CHAPTER V.

General Symptomatology.

THE symptoms of poliomyelitis are as varied as the extent of its pathologic lesions. They may be grouped under three general heads:—

1. The manifestations of an acute systemic infection.

2. The symptoms of an acute and diffused inflammation of the central nervous system.

3. Signs of acute and chronic localized lesions of cord and brain.

Incubation.—The period of incubation has not been definitely ascertained. It apparently varies with the virulence of the infection and the susceptibility and resistance of the patient. In experimental inoculation of apes, invasion commonly occurred in the second week, although the onset appeared as early as three days and as late as six months (Williams). Two to ten days are now accepted as the probable period in human subjects. A susceptible child whose resistance had been broken by unusual fatigue, exposed to repeated infections from hypervirulent sources, might present a very limited period of incubation, and die during systemic invasion. Such a case came under the writer's observation during a street fair in a western city. A child, coming from a healthy and uninvaded territory in the woods to the town where the epidemic was rife, was taken fairing all the first day and refused to go out the following morning. Onset and death followed in seventy-two hours.

Leegard, of Norway, who studied and reported the Bratsburg epidemic of 54 cases in 1899, considered the

period of incubation in some cases to have been less than twenty-four hours.

In some cases the incubation period has apparently lengthened to three weeks.

Aura of Attack.—In many cases there have been noted a series of initial symptoms before the actual onset of the disease. These prodromata, slight in themselves, are yet fairly constant, and so distinctive as to form valuable points in the diagnosis of the disease in this early stage. A change of disposition will be first noted, with irritability and peevishness. An unexplained malaise will occur, as in the case of the child who refused to go fairing. The child or adult may endeavor to ignore a weakness or prostration so unaccustomed. A feeling of sickness or shakiness will be experienced, attended with a slight dizziness or even vertigo. This vertigo and inco-ordination induce an ataxia which, taken with the accidents it is responsible for, form the aura of the attack.

The aura of the attack in a child is manifested by trembling, and a stumbling gait or unaccustomed falls. In the adult, owing to the better educated conduction paths between cord and brain, the stumbling is less frequent, but does often occur. The adult, however, will experience a slight mental confusion, a certain cervical tension, and unaccounted tremor and an undoubted ataxia. The large number of cases in both children and adults in which trauma is given as a cause of the paralysis are many of them cases in which the aura of the attack has been unmistakably manifest.

A history of trauma was given in 47 cases of 635 investigated by Dr. Lovett. Of these, 32 cases gave a history of accident followed almost immediately by paralysis, and the balance after an interval of some days. The tables given are of such interest in this connection that they are

included. It will be seen that only two of the falls can be excluded as not due to ataxia and inco-ordination,—child dropped by nurse; child hit by stone.

TRAUMA PRECEDING POLIOMYELITIS.

Tabulation of 47 cases, excerpt from Lovett.

Slight accidents:—	
Slight fall	20
Fall from cradle	3
Fall from swing	1
Fall from carriage	3
Fall from chair	1
Fall from automobile	1
Walking, skating or playing (fall)	5
Dropped by nurse	1
Falling under other children	1
More serious accidents:—	
Falling from third-story window	1
Falling from first-story window	1
Stone fell on head	1
Gate fell on foot	1
Fracture of tibia	1
Later cases:—	
Fall from carriage, chair and bed	3
Fall, paralysis three weeks later	1
Injury: Sprain of ankle	2

Dr. Gregor's Instances.—A child was at the wash-basin and fell while crossing the room for towels; a boy of 6 was observed falling about the house, went out, when he again fell and was carried home; a woman got out of bed and fell down, becoming dazed.

A fall down steps, down stairs, down a bank, or the grassy terrace surrounding a house, have all been mentioned to the writer as causes of the paralysis. The following case is typical of such histories, but the osteopathic deduction from the fall is as original as characteristic:—

Mary M., 5 years of age; slight fall two days before onset. Vomiting; pain in back and limbs; temperature 104° F., fourth day 100°, where it remained for two weeks or more. Paralysis fourth day; both lower extremities involved, left more than right; deltoids and shoulder group most affected; stiffness of neck and spine. Patient acquired almost perfect control of the arms and could use legs fairly well when last seen. Parents became dissatisfied with slow (?) progress of case and called an osteopath, who diagnosed condition as "fracture of the neck" due to fall two days before onset. (Dr. Pretts, Plattsville, Wis., August 1, 1908.)

Here the ataxia preceding more serious trouble is well illustrated:—

B. N., 4 years of age, male; predisposing cause autointoxication. Complained of pain in posterior surfaces of thigh. Walked like an old man, and also held to the wall for support, for two days. Stupor. Painful point over posterior branch of lower lumbar nerves. Paralysis: Both legs and muscles of erector spinæ group. July 30, 1908: Can sit up erect, extend right foot, flex and extend leg, and flex thigh. Left leg: Extends and flexes thigh, but cannot flex or extend foot. (Dr. G. H. Fellman, Milwaukee, April 29, 1908.)

A clear history of aura of onset in a case of adult poliomyelitis is given:—

G. G. H., aged 40; onset August 9, 1907. A certain degree of dizziness extending over two weeks preceded the onset of the disease. On August 9th, while at work, he began to suffer from a headache, which rapidly grew in intensity and became so severe that the patient spoke of it as the most agonizing pain he had ever experienced and should never forget. Prostration, high fever, vomiting, appeared and continued for four days; on the fifth day of onset the patient had totally lost the use of his left arm; during the sixth, seventh and eighth day the two lower extremities, the trunk muscles and the muscles which support the head were involved in the paralysis, with temporary paralysis of the bladder, and paresis of the right shoulder muscles. [Abstracted from Archambault's "Poliomyelitis in the Adult" (Albany), N. Y. Medical Journal, August 8, 1908.]

This man of 40 gives a distinct history of dizziness lasting for two weeks preceding the terrific onset of the disease, which also continued for ninety-six hours before the character of the attack was clearly defined by the oncoming paralysis. Adults are better able to give a lucid account of the character of their symptoms than children; and if the subject of poliomyelitis in the adult had not been almost wholly overlooked or ignored, we could doubtless present a series of symptoms so uniformly present as to make diagnosis of the preparalytic stage of poliomyelitis in the adult a definite entity.

Onset.—The symptoms of onset are also the symptoms of the arrested or so-called aborted form of the disease. The onset is cumulative rather than abrupt; the history of an absolutely abrupt onset of this disease is due to the fact that slight febrile conditions are often overlooked in children and ignored in adults. The seeming suddenness of onset is due to the simultaneous rise in temperature, pulse and respirations, together with meningism, basilar headache, tremor, convulsive movements, and vomiting, with obstipation, and retained or suppressed urine. The onset occurs very often in sleep, the patient waking with high fever, and all the other symptoms enumerated. It may begin with a chill; it occasionally begins insidiously, with the graduated approach characteristic of typhoid fever.

A marked increase in the pulse rate to 140-150 is perhaps the most constant feature of onset. This is accompanied by a sharp rise in temperature to 104-106° F. Respirations are rapid, increasing to 40 or 60 per minute with no apparent cause. An immediate paresis of digestion is evidenced by severe and repeated vomiting, and constipation. Inco-ordination, tremor and ataxia are increased, and prostration is marked. Occipital headache

and pain of an agonizing character between the shoulders and in the lumbar region are usually present. Cervical tension or rigidity will be complained of, or the whole spine may be spastic. Sleep is broken by muscular twitchings, which vary from a light jerking of the extremities to convulsive movements violent enough to throw the patient from the bed. Urine is scanty, retention frequent, and suppression not infrequent.

The type of onset varies somewhat in epidemics; meningism is the most marked feature at times, while gastroenteritis was more prominent in the Germanic epidemics of 1908-9. Some cases begin with an angina simulating tonsillitis, and in every epidemic a small percentage of cases develop with an acute multiple neuritis.

Circulatory System.—Epistaxis occurred during the onset in a number of the Wisconsin cases. Nasal hemorrhage may have been due to an individual susceptibility; it is more probable, however, that an extreme congestion of the nasal mucosa is excited, which is conducive to hemorrhage. We know that the nasal and pharyngeal mucosa and tonsillar tissues provide an atrium for the infectious virus. We also know that the walls of blood-vessels of the cord are rendered pervious by the action of this virus, and we suspect that the virus in some instances travels by way of the nasal mucosa and cribriform spaces of the ethmoid directly to the cerebral mucosa. It would appear that a congestion of the nasal mucosa, destruction of the integrity of the wall of the nasal vessels, and a concurrent epistaxis may accompany the onset. Epistaxis at the onset occurred frequently during the Cornwall (England) epidemic of 1911.

The heart is much accelerated, often to double the usual rate. In the cases among children which fell under the writer's personal observation, the pulse during the

acute stage ranged from 160 up, being counted with difficulty beyond that point. Dakin, in his report of the Mason City cases, states that the pulse is usually high, 100 to 150 in adults and ranging up to 200 in children. In a majority of cases the child is not seen until this stage is passed and paralysis has supervened, when the pulse im-

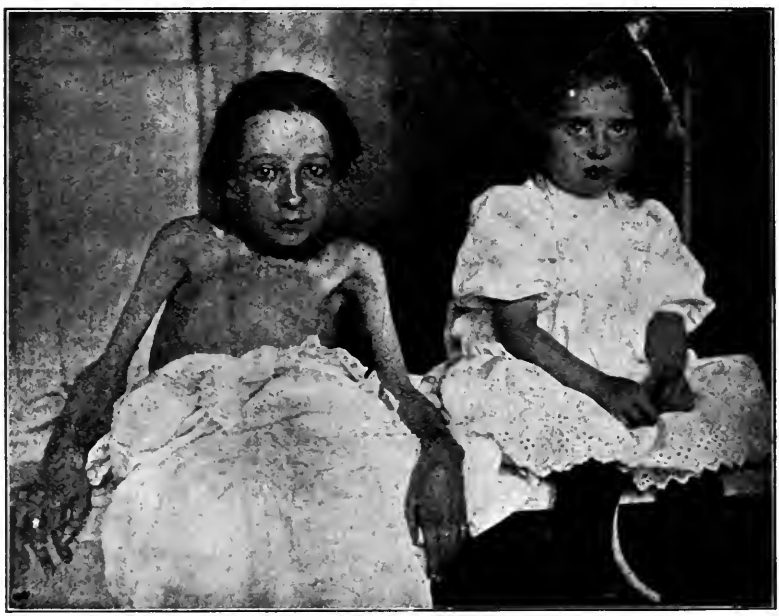


Fig. 33.—Aunt, 18 years of age, poliomyelitis at 9 years of age, and niece, a normal child, 3 years of age; paralysis, atrophy, contractures and trophic non-development. (From series of *Dr. Louis Ager,* of Brooklyn.)

mediately declines to about 100. The fall to normal may be delayed until invasion is over and a recession of the paralysis is evident. The pulse is weak and compressible in character, and may show marked irregularity. This early and violent change in the heart action is probably due to irritation of the accelerator branches of the vagus. It may be caused by the virus in the blood acting on the

center for these accelerator fibers, as it occurs before there is any evidence of destructive lesions in that area. This acceleration, however, may be due to the direct irritant of the virus on the heart muscle; such was considered by Rekseh, of Rhenish Westphalia, who reported the following case:—

A girl aged 8, after a mild prodromal stage, developed a flaccid palsy of both legs. Her general condition was excellent, and all of

Fig. 34.—Posterior view of Fig. 33.

the organs, especially the heart, appeared healthy, when, without any warning, death occurred suddenly on the second day, being probably due to the action of toxins on the heart muscle.

The increase in the pulse rate may be independent of temperature. The pulse rate is often unmentioned in otherwise complete reports, or it is manifest that the pulse recorded was taken in the later paralytic stage. The complete report given by Frizzell of the Princeton student's case is indicative of the typical cardiac change. It is stated that this patient was seen fifty-six hours after the first

intimation of illness, with a pulse of 90. The evening of that day it reached 120. The day was October 31st; Nov. 1st, the pulse declined to 90; Nov. 2d, to 80 beats, and Nov. 3d, when paralysis appeared, the pulse declined to 60-64, near the normal rate for a young adult male with a sound cardia.

A decrease in the pulse rate occurs in the postfebrile drop, and in cases of the sudden fulminating type. Persistent headaches, and a subnormal pulse and temperature followed an attack of the arrested type in an adult for a period of eight weeks. A pulse rate of 42 was reported by Dr. Gregor and Dr. Hopper, during the Cornwall (England) epidemic:—

A. J., male, aged 25. A perfectly healthy man up to the time of his fatal illness; no history of tubercle; Sept. 22d, severe frontal headache; retching; rose at 11 A.M., went for a short voyage on the bay; on the way home became sick, and vomited (not seasickness). Arrived at dock at 5 P.M. Too ill to walk home, but walked up steps to a cab. 5.30 P.M., pulse 42; curled up in very lethargic state; feet cold; did not speak, but put out tongue when told; vomited a brown, grumous fluid; became comatose; died at 8 o'clock P.M. *Post mortem:* All organs healthy except brain; meninges much congested. Lateral ventricles distended with fluid. Encephaloid type.

Vasomotor control is disorganized. Sweating is frequent and in some epidemics of such a profuse character and so constant that it is spoken of as a cardinal symptom of the disease. The conjunctiva may be congested. The face assumes a mask of brilliant red and white. Later, the paralyzed extremities become ecchymosed, bluish in appearance, and cold to the touch.

Temperature.—A chilly sensation sometimes accompanies the onset in the adult and more rarely a distinct chill occurs. It is probable that this occurs not infrequently among young children, being overlooked or taking the

form of a slight convulsion, which is the way a rigor is usually manifest in infancy.

At the onset of the attack the temperature jumps to or near the maximum point attained. The temperature is high, rising to 104-106° F. for a short period of time, when it declines by crisis to about 100°, except in the most serious cases. It may rise higher, but 105° is the average elevation. The temperature is variable and may be misleading if this initial rise has not been noted. The temperature is not a reliable indicator of the severity of the attack, but a high temperature with correspondingly severe onset usually indicates extensive impending paralysis. The temperature declines by crisis before the paralysis is manifest in the majority of cases. It remains at or near 100° while the paralysis is extending, and then drops to subnormal in uncomplicated cases. The surface temperature of the affected parts is depressed.

The burning fever of onset is sometimes found unendurable by the victim. R. V., a 10-year-old boy reported by Marquardt, of La Crosse, sought a garden sprinkler and laid under it, developing a paralysis of all four extremities. His temperature when seen was 102°. Its height at onset can only be estimated. There is undoubtedly grave irritation of the heat centers in a majority of cases.

The temperature and symptoms are not infrequently mistaken for heat stroke:—

> A child dying in a paralytic or comatose condition might have been regarded as a case of sunstroke. One such example occurred in Devonshire, where two children were supposed to have died of sunstroke, whilst a child who had been staying in the same house developed poliomyelitis immediately on its return to Plymouth. It is more than likely that both the fatal cases were infected with poliomyelitis, more especially as they were young children who had not been exposed to the sun. (Soltau, Brit. Med. Jour., Nov. 4, 1911.)

The temperature of the cases of the arrested or so-called abortive forms is not always secured. The mother will say that the child had a high fever for one night only. Such a case is given where the temperature was secured:—

H. A., male aged 7, residing on a farm five miles away. Onset sudden, July 1, 1910. July 2d, temperature 102.5° F.; severe occipital headache; pain and tenderness of spine. July 3d, temperature normal; pain and tenderness had disappeared. Two other children in same house were ill, June 29th and July 1st, with similar symptoms. One recovered in a few days; the other developed typical poliomyelitic paralysis of the arm. (Dr. Fred. Albert, Mason City, Ia.)

The arrested type may show as high a temperature as serious or fatal cases. The fever in complicated cases may be continuous or rise to the same point as that of sunstroke. Dr. Fellman, of Milwaukee, reported a case of the cerebral type, which developed a temperature of 108.5° to 110°, per rectum, in the fourth week. The case was fatal.

Respiratory System.—Sneezing is a frequent early symptom. Coryza is so rare an accompaniment of this disease that its presence at the onset is probably coincidental only. A pharyngeal angina occurs with more frequency; the tonsillar tissue has been found infected with the virus, which would account for pharyngeal and tonsillar congestion.

Respiration is profoundly affected in cases with a severe onset or extensive lesions. Respirations are at first rapid, rising to 40 or 60 per minute with no apparent cause. This rise may be attributed to the irritation of the phrenic nerve at its common origin with the spinal branches of the spinal accessory. A marked increase of the number of respirations per minute, due to irritation of the phrenic supply of the diaphragm, occurs with cervical tension and

hyperextension of head due to spasticity of muscles supplied by cervical plexus. (Spinal accessory and second, third and fourth cervical.) The lungs are found clear. Rapid respiration does not occur in cases of a very mild type, nor is it seen in cases of the arrested type with a mild onset. It reaches normal with the lowering of the fever and the appearance of paralysis. Two cases are given, one of the arrested type, one of the paralytic, both with very rapid respirations at onset:—

No. 10. J. L., 6 years; direct exposure; sudden onset; chill; vomiting; pulse 150; temperature 102.4°; respiration 60; no paralysis; recovered.

No. 24. K., 1 year; indirect exposure; sudden onset; vomiting; pulse 180; temperature 104°; respiration 80; muscular twitching; prostration; sweating; spasticity; paralysis of anterior tibial; contractures; improving. (Dakin's series.)

Should the case prove to be one of the acute ascending type of paralysis, the respirations will remain elevated, or after a brief drop will again accelerate, becoming feeble in quality and somewhat irregular. This second or delayed involvement is due to a beginning paralysis of the muscles of respiration, the chest walls remaining fixed and the breathing assuming the abdominal type. The diaphragm may be paralyzed.

With diaphragmatic paralysis the upper abdomen recedes during inspiration and is protruded during expiration. Such a case may present a mild paresis only, a normal temperature, and be fully conscious, when sudden paralysis of respiration and death occur.

Dyspnea is marked, and the acceleration of the heart shows involvement of the vagus. The Cheyne-Stokes syndrome may occur in both types of paralytic involvement of respiration.

The muscles of one side of the chest may be paralyzed,

and the chest immobile, while the respirations are regular but feeble, and the respiratory excursion of the other half of the chest still evident. This type of breathing is paralytic. It is a bad symptom, but cases have been known to recover.

A very rapid paralysis of respiration is due to bulbar involvement. Such a case may present a mild paresis only, a normal temperature, and be fully conscious, when sudden paralysis of respiration and death occurs. Cheyne-Stokes syndrome indicates involvement of the vagus.

Digestive Tract.—As has been well said, there is an immediate paresis of digestion with the onset of poliomyelitis. It is evidenced by anorexia, vomiting, foul breath, sordes, and a disordered elimination which may take the form of diarrhea, but in the vast majority of cases it is manifested by a stubborn constipation with colic, tympany and meteorism.

Lack of appetite accompanies the prodromal stage; nausea and retching may or may not precede the vomiting. Vomiting is sudden and repeated. It may be constant for two or more days, and is sometimes of the violent and projectile type. The vomiting is often attributed to faulty digestion or an indiscreet dietary (green apples, sausage, sauer kraut). The outraged stomach disgorges food it is unable to assimilate, but repeated vomiting after evacuation is due to irritation, probably at the center. Projectile vomiting may be so violent that the vomitus stains the wall or ceiling of the patient's room. Wickmann reports a case of a woman vomiting with such force as to dislocate her jaw.

The vomiting of a brown, grumous material is reported as occurring late in many fatal cases. The coffee-ground appearance of partly digested blood is suggested by these reports, and may result from an epistaxis with swallowing

of the blood by the unconscious patient; exact knowledge on this point is to be acquired. Vomiting of the ordinary type was present in 62 of the 150 tabulated Wisconsin cases, of which 1 case is given:—

L. S., male, aged 5 years; pain; vomiting; marked constipation; prostration; nervous and irritable; temperature 104°; rash; convulsive; both heels drawn back upon buttocks; sensory nerves very acute in beginning of case, slight handling producing severe pain; paralysis of both legs. Oct. 6th, unable to sit alone; legs lie extended, recovering motion in thigh muscles. (Dr. G. W. Menika, Readstown, Wis., Sept. 20, 1908.)

It is to be noted in this case that vomiting and constipation were present at the onset, but in the report the emphasis is put on the constipation. The checking of the elimination in these cases, which is most frankly manifested by a stubborn paresis of the bowels, should not be ignored. It is a constant symptom; it is a dangerous condition; in its detection and relief lies our only present hope of lessening or preventing the oncoming paralysis. It is interesting and suggestive to note that in experimental inoculation of monkeys the virus introduced into the digestive tract proved inactive unless peristalsis was artificially inhibited.

Constipation is present in a vast majority of cases. Diarrhea is the pre-existing condition to this fecal stagnation in some cases, and denotes an effort of the digestive tube to cast off the virus. In this country diarrhea is most often noted in the arrested or abortive type of case; the concurrent diarrhea noted in other members of the family during the progress of a case is a very certain indication that they have taken the infection. Diarrhea was reported as a more constant symptom in the Westphalia epidemic of 1908 (Krause). The bowel movements are large. The fecal stagnation is due to a temporary paresis of the tract; it is very resistant to catharsis and colonic flushings. In

the carefully watched case of the Princeton student the bowels "were moved only with great difficulty for a month" from the time of onset, and this case was so mild that it just escapes classification as one of the arrested type.

The bowel movements are foul until free defecation is established; thereafter the stool appears to be normal.

The tongue is red at first, the papillæ of the anterior half noticeably dilated and scarlet; it subsequently becomes coated, and sordes gathers on the teeth and gums.

Genitourinary System.—The urine is scanty and high colored. Albumin is not constant, but is found in a certain percentage of cases. An acute exudative nephritis has been reported. Frequent micturition occurs early, the symptom of a mild cystitis due to elimination of the virus by the kidneys. Retention or suppression may follow. Retention of the urine is a common occurrence. It is stated to have occurred 11 times in 150 Wisconsin cases, but it is often overlooked. Retention is due to a temporary paresis of the walls of the bladder, together with scanty secretion. When the paralysis extends to the urinary sphincter there is incontinence; this is somewhat rare, and always temporary in uncomplicated cases. Delayed urination and dysuria may occur. Anuria and suppression have been noted in rapidly fatal cases of the spinal type. Retention and overflow may occur. Scanty secretion and retention are features of the first of the following cases; retention and incontinence of the second:—

Princeton student, 21 years; onset Oct. 29, 1910; Oct. 31st, pulse 90 to 120; temperature 101°; respiration 22; last urination Nov. 2d, 6.30 P.M.; Nov. 3d, unable to urinate; lumbar paralysis same day. Catheterized, scanty flow of urine; pressure over symphysis used; bladder paralysis lasted for twelve days; bowels were moved only with great difficulty for a month. (Dr. Frizzell, N. Y.)

CASE XVI.—D. G., age 2½ years; acute onset August 8th; vomiting, retraction of head, retention of urine and distended

bladder, with diagnosis of intussusception of bowel. Removed to hospital and operated; nothing found but an excessively distended bladder. August 18th, paralysis of both legs and right arm cleared diagnosis. (Reported by Lovett and Jones, Bulletin Mass. State Board of Health, June, 1910.)

C. T., male, 1 year 6 months; onset August 21, 1911; drowsy, febrile; clonic spasm; opisthotonus. Paralysis: August 23d, all four limbs and back; retention of urine and overflow; death August 28th. (Dr. Moss-Blundell, Huntingdonshire.)

With an irritant and destructive process in the lumbar and sacral cord, and frequent involvement of the bladder, it is doubtful that the genital organs wholly escape functional and organic alteration. There seems to be little observation recorded on this point. The following case is the only one included in the Wisconsin report:—

W. B., male, 6 years; onset September 26, 1908; chill, temperature 103°; rapid pulse; headache; delirium; apathy; stupor; rigidity of neck; dyspnea; pain and tenderness over the whole body; photophobia; constipation; semierected penis all the time; Kernig's sign present. Paralysis: upper part of back and right and left arm and forearm, but not hands. (Dr. L. A. Larsen, Colfax, Wis.)

A male adult presenting an incomplete transverse myelitis with acute onset had great pain in the testicles, followed by slight paralysis of the quadratus femoris. With a diagnosis of probable poliomyelitis the spinal fluid from this case was injected into a monkey, which became paralyzed six months subsequently. (Williams: Discussion, Section of Medicine, Penna. Medical Society. Penna. Medical Journal, December, 1911.)

Cutaneous System.—Sweating may be profuse and has been reported as a characteristic symptom in some outbreaks. It is not constant.

Cutaneous hyperesthesia is common; it may take the form of a subjective sensation, the child complaining that there are flies walking across the skin, etc. One little girl said the mosquitoes were killing her. The cutaneous temperature sense may be exalted or depressed; a hot-

water bag may be agreeable to the paralyzed leg and unsupportable to its fellow. Tactile sensation may be delayed or exalted.

Vasomotor changes of the cutaneous surface occur in the paretic member, which is congested in appearance, with a lowered temperature.

A skin rash is found in something more than 10 per cent. of the cases and is often overlooked. The rash is multiform in character; it may be erythematous or urticarial in appearance, morbilliform, petechial, papular, pustular, or purpuric.

The measles-like rash is most common; it frequently leads to a diagnosis of measles. It consists of patches, not so large as the typical measles blotch, but otherwise resembling it closely. It does not, however, appear on the face and at the hair line, as in the classical measles rash, but is found first on the torso, spreading from there to the extremities, and to the dorsum of the feet.

All of these varieties of rash were seen in the Wisconsin epidemic. A well-developed scarlatinal erythema occurred in a child of 6 years with paraplegia and constipation. A fine pustular rash was seen in 2 fatal cases in brothers, covering the torso of one and barely noticeable in the other; the rash was fine but distinctly pustular. Purpura appeared on the hips and thighs of a rapidly fatal case.

S. W., 4 years, male; onset August 4, 1908, with pain, vomiting, delirium and twitching, but no convulsions; temperature high; paralysis of both lower and both upper extremities, trunk and neck muscles; rash resembled measles, very intense; atrophy. (Dr. A. W. Myers, Milwaukee.)

N. O., male, 2 years; onset October 10, 1908, with vomiting and diarrhea; fever; small, pimply rash about trunk and neck; paralysis of both lower, atrophy of left, recovery of right extremities. (Dr. C. E. Armstrong, Oconto, Wis.)

Dr. Anderson, of Nebraska, reported a rash often seen in their

epidemic as characterized by rose-colored spots from one-half to two inches in diameter, which faded to brown and disappeared.

An urticarial rash was present in 6 cases of poliomyelitis which were seen through all the stages of the disease in the Cornwall, England, epidemic of 1911. It was recorded as appearing three times on the abdomen and once on the abdomen and buttocks. (Gregor and Hopper.)

The multiform character of these rashes is evidence, as Frost has reasonably stated, that "no skin eruption can be said to be at all characteristic of acute poliomyelitis;" yet the virus, acting with malignant energy on every part of the system, induces a frequent cutaneous reaction, which may simulate any of the eruptive diseases. It should be borne in mind that such rashes may occur in poliomyelitis, to avoid confusing this disease with the relatively harmless acute eruptive fevers.

Labial herpes was reported once in Wisconsin. Wickmann considers the condition not characteristic. It may be coincidental. Brown, of Toronto, reported a skin eruption in 6 consecutive cases of poliomyelitis, which followed the same course in each case. In each of the 6 cases a vesicular eruption was present when the case was first seen. The rash was typical, being present more or less all over the body. It was papular and vesicular, and was present also on the lower extremities, on both anterior and posterior surfaces. Sections through the vesicles show the latter not to be deep seated, as one would imagine on palpating them, but, on the contrary, to be only superficial, *i.e.*, between the Malpighian and corneous layers of the skin. Apart from the perivascular infiltration no other pathologic change could be found. Smears and cultures from the serum of the vesicles gave no uniform result.

Mental State.—A feeling of grave apprehension accompanies the onset of poliomyelitis, which has been ex-

pressed as a feeling of impending danger. A farmer said he felt as if a cyclone was coming, and knew he must get the crops in, working into the second day of onset before he gave up. Prostration is early and more extreme than the symptoms would at first indicate. The profound effect of the virus acting on the ganglionic axis may provoke this unconditional surrender, but it may be regarded as a protective defense of nature. Evidence favors the theory that the patient who surrenders immediately lessens the danger of paralysis with the recuperative power of rest. Ball, of Minnesota, notes, in the series of cases he investigated, that in the fatal cases the patients took a varying degree of exercise after the onset of the disease.

A staring and frightened expression is often seen. Dakin, of Iowa, considered this facies characteristic enough to be called pathognomonic. "The face appears drawn; the eyes sunken, with contracted pupils, staring in an unmistakable, unwinking terror. The expression of fear has been noted by the families of patients in almost every case." The facial expression is noticeably anxious, questioning, and gives a prematurely aged cast to the countenance.

Excessive emotionalism, hysterical laughing and crying, and change in disposition of children have been noted among the early symptoms. The adult may be extremely restless, and conscious of a vague anxiety and mental confusion. In this state and driven by apprehension, he may continue to automatically perform his work for some time after the onset. Mental confusion and determination to remain at work are shown in the following case, with its speedy lethal close:—

E., aged 24; single; engineer; admitted at 12 noon; weakness; pain in right hip; inability to use hands; seven days previous he developed a sore throat, but worked until the second day before

admission; went to bed; during these seven days there was headache, pain in the neck; he had vomited. was constipated, and reported difficulty in thinking. On admission: pulse 76; temperature 99°; could not hold a cup in his hands; hands semiflexed; breathing abdominal and jerky.

Seven P.M. same day: respiration 65; temperature 99°; pulse 56. In spite of extreme dyspnea, accessory muscles of respiration were not in action. 12 midnight: cerebration apparently clear. 3 A.M.: death, sudden, of respiratory failure. (Dr. Colin Russell, Royal Victorian Hospital, Montreal.)

Pain.—Pain is a constant and early symptom of poliomyelitis. Its usual occurrence is in the form of basilar or occipital headache; this may be associated with an agonizing pain between the shoulders and in the lumbar region of the spine. In rare cases the headache is at first frontal; it then localizes in the occiput or at the cervical nuchæ. There may be a racking pain of the entire spine, which the patient will speak of as a backache. "I have had backaches before, but never anything like this!" said a farmer of 50 years, during the onset of the disease which proved to be fatal. Occipital, cervical and spinal pains are most characteristic of this disease, indicating their origin in the acute inflammation of the meninges and spinal ganglia.

Myalgia of a mild or severe grade attacks the segments that will shortly become paralyzed; the myalgia of an extremity which precedes and accompanies the paralysis of that area is not to be confused with the neuritis of great nerve-trunks of the so-called neuritic types of the disease. The most careful handling of these cases will cause agony, but it seems possible to differentiate slightly, *i.e.*, that pressure more than movement causes the pain. The pressure pain is shown by the child submitting to be lifted to a chamber, but screaming with pain when resting on it. Myalgic pain may be constant or paroxysmal; it invariably precedes the paralysis of the segment or group of muscles,

and may cease with the oncoming of the paralysis; it more frequently remains for a varying period after the onset of paralysis, and may continue for weeks or months. Wickmann considers that the extreme pain on movement is due to tenderness and rigidity of the spinal column.

Peripheral neuritis may characterize another class of cases, with hyperesthesia and an intractable pain along the nerve-trunks and into the posterior root ganglia. These cases do not usually develop a paralysis, but the pain and tenderness of the nerve-trunk may be long continued. An adult patient in whom the disease was of fifteen years' standing had never been free from pain for a greater period than a day. Pain is a minor feature of one class of cases. The rapidly fatal cases of the bulbar type are very quiet, and do not seem to suffer.

Pain and Tenderness.—The cases investigated indicate that pain or tenderness was present at some stage in a great majority of cases, and it is well to emphasize its importance among the few really reliable early signs:—

Pain and tenderness was present in.....	98	cases.
Pain and tenderness was absent in.....	25	cases.
Pain and tenderness was not stated in....	13	cases.
	136	cases.
Doubtful or abortive cases.....	10	
	146	cases.

In this connection it is interesting to note the length of time in which the pain and tenderness lasted, as is shown by the following table:—

One day or less	1 case.
Two days	4 cases.
Three days	4 cases.
Four days	9 cases.
Five days	1 case.
One week	8 cases.
One to two weeks	8 cases.
Two to three weeks	21 cases.
Three to four weeks	7 cases.
Four to five weeks	15 cases.
Five to seven weeks	4 cases.
Seven to eight weeks	1 case.
Eight to nine weeks	4 cases.
Still persisting	7 cases.
A few days	4 cases.
No pain or tenderness	25 cases.
Not stated	13 cases.
	136 cases.
Doubtful or abortive cases	10
	146 cases.

(Kelly, State of Washington.)

Meningism.—Symptoms of meningeal irritation are rarely absent and in the meningeal type of the disease are marked. Cervical pain and rigidity, with some retraction of the head, are commonly seen. The spastic condition may vary from a slight stiffness of the neck which prevents flexing the head on the chest, to retraction of the head, or an opisthotonus of so severe a degree that the child lies like a bent bow. The spasticity of the muscles may give a rigid spine with no flexing of same; "stiff as a board," "stiff as a log from head to heels," are not exaggerations of the condition in some of these cases. According to Frost, this stiffness of the spine may be due to the pain and only in rare cases is due to actual contracture of the spinal muscles.

Reflexes.—The patellar reflex, when ascertained, may be misleading. It is usually exaggerated during the early stage, and often unobserved. It is diminished or disappears prior to the onset of the paralysis in the usual case. As the paralysis of one leg usually precedes the paralysis of its fellow by twenty-four to forty-eight hours, the reflex may be exaggerated in the one extremity and abolished in the other. There may be a persistently exaggerated patellar reflex in paralysis which affects only the upper segment. Spastic cases are unable to completely extend the leg when the thigh is flexed at a right angle; this is known as the modified Kernig test, and is not usually present until the second day. Dakin notes a characteristic sign of stiff neck. The patient, if asked to touch his chin and chest, will endeavor to do so by opening the mouth and depressing the lower jaw, the neck remaining rigid. In cases of cervical rigidity with involvement of the sternomastoids, the patient, if told to look at an object, will roll the eyes but make no attempt to move his head.

The superficial and deep reflexes vary widely in reaction in the different types and stages of this disease, and have not been satisfactorily classified in regard to it. The ocular reflexes are not characteristic, being subject to disturbances.

Meningitis.—As the irritant stage continues the symptoms of meningeal involvement become more grave. Twitching and jerking of muscles and tremor appear. Convulsive movements may be manifest as a twisting of torso, or true convulsive seizures occur. The dismissal of this subject with a brief note that convulsions sometimes occur in childhood is as misleading as the name infantile paralysis. Convulsions of the most serious and rapidly fatal type may occur at any age and to members of both sexes. The virulence of the infection and the area of the

nervous system invaded are the factors that determine the convulsive seizure, and not age nor sex. The only evidence of the disease in child or adult may be a convulsive attack of short duration. The attack may occur under the most misleading circumstances, and may simulate, and be diagnosed as any of the diseases characterized specifically by convulsive attacks, notably eclampsia, tetanus, or rabies:

H. K., female, 27; sudden onset August 19, 1906; fever, headache, pains in back; stiff neck; retracted head; violent tonic contraction of shoulder muscles, forearms, arms and hands; cramps so painful as to require chloroform; cramps continued second day and night, opisthotonus. August 21st: the patient being six months pregnant, eclampsia was suspected and forced delivery undertaken successfully. Cramps continued, extending to legs; dysphagia and aphonia occurred; death at 6 A.M., August 22d. Conscious throughout; autopsy revealed typical lesions of poliomyelitis. (Wickmann.)

Confusion, apathy, stupor, and delirium are all seen, and of a transitory nature in most cases. Coma is rare:—

September 25, 1908, E. T., male, aged 8 years. Headache, pain in back and neck; temperature 102° F., comatose for several days; no paralysis; recovery; meningeal type, no spinal symptoms. (Dr. Bowles, Eleva, Wis.)

General Features of Acute Attack—Symptoms.—198 cases give history of fever; 184, pain and tenderness; 117, brain symptoms; 106, headache; 79, retraction; 59, sore throat; 24, apathy; 19, delirium; 18, rigidity of neck; 13, cough; 13, irritableness; 11, restlessness; 11, unconsciousness; 9, tired condition; 8, rigidity of spine; 8, lassitude; 7, strabismus; 6, change of temperament; 6, diaphragmatic breathing; 6, dysphagia; 5, sweating; 5, irregular pulse and respiration; 5, twitchings; 5, diminished or absent reflexes; 4, convulsions; 4, nystagmus; 3, weakness; 3, hyperesthesia; 3, stupor; 3, exaggerated reflexes; 3, regurgitation of food; 3, anorexia; 1, thirst; 1, anxious expression; 1, tremor; 1, coma; 1, tympanites; 1, insomnia; 1, hiccough. (Sheppard.)

CHAPTER VI.

Symptomatology of Special Types of Acute Poliomyelitis.

RECLASSIFICATION OF TYPES OF POLIOMYELITIS.[1]

I. *The Arrested Type.* (Most frequent. Abortive: old classification.)

II. *The Spinal Myelitic Type.* (Flaccid paralysis.)

III. *Acute Ascending or Descending Spinal Paralysis.* (Landry's. Separation from Type II arbitrary. Both spinal origin.)

IV. *Acute Bulbar.* (Cranial nerve paralysis: facial, auditory, hypoglossal, oculomotor.)

V. *Encephalic Type.*—Polioencephalitis superior: Rolandic cortex—spastic hemiplegias. Frontal area—mental defectives. Occipital area—blindness with normal eye-grounds.

Polioencephalitis inferior: Acute bulbar-pontine type: see above.

Encephalitis cerebelli. The predominant *acute ataxia type.*

Encephalitis of midbrain and connections. *Acute tremor.*

Thalamic encephalitis. Spastic para- or hemi- plegias associated with athetoid and choreic movements and tetany.

VI. *Meningitic Type.*—With or without paralysis.

[1] Modified from Wickmann and Reginald Miller, to whom we are indebted for much recent knowledge of the disease.

VII. *Neural Type.*—Acute multiple neuritis. Sciatica. Herpes zoster. Chorea.

VIII. *Rapidly Fatal Institutional Disease Type.*

There may be no marked distinction between the types of poliomyelitis; the cerebral type may present symptoms

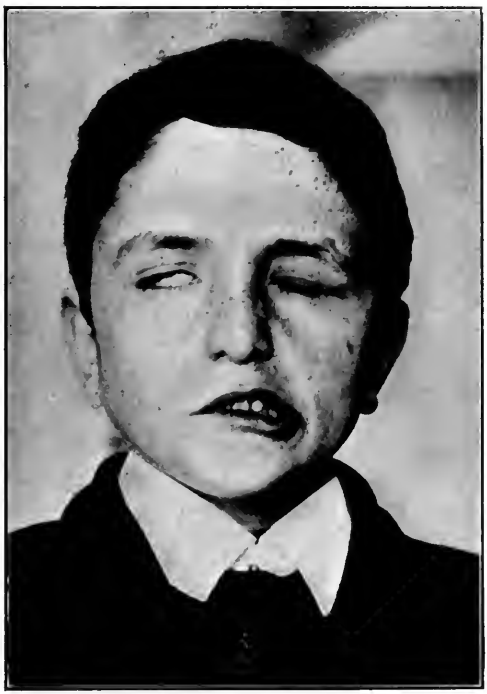


Fig. 35.—Paralysis of facial nerve.

of spinal involvement and *vice versâ;* the localization of this disease may exhibit every caprice of selection.

The symptom relationship between all types is shown most clearly during the earliest stages of the disease, for all types present a similar onset. This symptom group of onset is also the clinical expression of the most numerous class of cases, the arrested, formerly called the abortive, type of this disease. Next in frequency appears the typical

form of the disease known as infantile paralysis, characterized by a flaccid and regressive paralysis of muscles supplied by spinal nerves. Every epidemic also presents cases where most pronounced symptoms indicate meningeal, pontine, cerebellar or cerebral involvement. Some hesitation has been shown in ascribing peripheral neuritis, whether multiple in character or of single neuron involvement, to the same cause. Clinical evidence of such relationship has accumulated, and pathologic confirmation is confidently expected.

I. THE ARRESTED TYPE.

The arrested form of poliomyelitis is given first in the reclassification of the types of this disease for the following reasons:—

1. The arrested form occurs more frequently than any other form, probably more often than all others.

2. Transmission of poliomyelitis may be largely due to frequent undiagnosed cases of the arrested type.

The change in the name of this type of poliomyelitis from abortive to arrested was determined on for the following reasons:—

Ignorant of the exact method of transmission of this disease, and lacking an antitoxin for prophylaxis and cure, it is certain that the epidemic will be checked in but one of two ways:—

(*a*) By the exhaustion of the material on which it feeds.

(*b*) By enlisting the intelligent co-operation of the public to aid in the detection and regulation of every focus in which it has located, but to insure intelligent attention we should not use misleading terms. The word abortive is clear to the medical man, for its adverbial medical meaning is "tending to shorten in course." To the laity in this

connection the word is dangerously misleading, as its common adverbial meaning is "coming to naught." Miller's term "rudimentary poliomyelitis" is better, and yet lacks somewhat of precise definition.

The arrested case of poliomyelitis may transmit the infection to those with whom he comes in contact; he may himself develop within a longer or shorter period the typical paralytic form of the disease. In other words this individual has the disease, but owing to some fortunate bodily resistance the disease is arrested. If fatigue or exposure lessens the resistance, the disease may proceed to any one of several terminations. For these reasons we recommend and employ the use of the definite and true descriptive adverb arrested to the most common type of poliomyelitis, rather than the misleading term abortive.

The following case is a good example of the arrested type in which a correct diagnosis would have been impossible except for association with the frank case of the child's brother:—

109. *Mild Anterior Poliomyelitis.*—There are several interesting points in Bullard's case. A child 8 years old had been thoroughly exposed to contagion from her brother, ill with anterior poliomyelitis. The patient developed an illness exactly one week later than her brother. Her illness, although the high temperature lasted but a short time, was a severe one, weakening the patient, who was a vigorous child, and at the time of the attack and for some time previously had been in perfect health, so that she was in bed fourteen days and was still weak at the end of three weeks. There was in this attack *no important* gastrointestinal disturbance. The patient was for two days unwilling to eat and there was *constipation.* Once when pressed to eat she vomited, but this was evidently due to a temporary condition, and was a secondary symptom. The general hypersensitiveness of the nervous system was very marked. The absence of paralysis is a significant feature. The brother had a typical case of anterior poliomyelitis with paralysis at first of all the limbs; at the present time paralysis of the

Fig. 36.—Typical case of poliomyelitis, lateral view.

lower extremities remains. (Bullard, Boston Med. and Surg. Journal.)

The proof of the existence of the arrested form of poliomyelitis has been summarized by Frost as follows:—

Etiologic Identity of Abortive (Arrested) and Paralytic Forms.—(*a*) Cases presenting the same initial

Fig. 37.—Same as Fig. 36, anterior view.

Fig. 38.—Same as Fig. 36, posterior view.

symptoms as paralytic cases occur coincidently in epidemics and recover in a short time without paralysis.

(*b*) Every closely studied epidemic shows a gradation in severity of nervous symptoms; extensive permanent paralysis; slight transient paralysis; partial paralysis

(paresis); ataxia without paralysis; meningitic or neuritic symptoms without motor disturbance; general infection without distinctive nervous symptoms of any kind (basilar headache and cervical tension always present). A group of cases showing all these gradations, occurring within a circumscribed area within a short time, all presenting somewhat similar initial symptoms seldom fails to convince the observer of the existence of abortive (arrested) cases of poliomyelitis.

(*c*) The occasional occurrence of such cases during an epidemic of poliomyelitis might be put down to merely coincidental prevalence of two or more distinct infections; the frequent, almost constant occurrence of such cases in intimate association with frank cases of poliomyelitis cannot be ascribed to fortuitous coincidence.

(*d*) Experiments have demonstrated that monkeys inoculated with poliomyelitis occasionally develop an abortive form of the infection, characterized by rather mild and indefinite symptoms. Roemer and Joseph have demonstrated in monkeys an immunity following such abortive attacks.

(*e*) Netter and Levaditi have shown that the serum of a child recently recovered from an abortive attack (arrested type) was capable of neutralizing the virus of poliomyelitis. It is, therefore, well established by clinical and experimental evidence that the infection of acute anterior poliomyelitis may cause slight illness without definite motor symptoms.

Frequency of the Arrested Form of Poliomyelitis.—Recent extensive study of epidemics of poliomyelitis in small communities have made it evident that the arrested form of the disease occurs as frequently as any other type, and perhaps more often than all other types taken together. In the careful investigation of the following epidemic in a

school district in Iowa, 5 cases out of every 6 were of the arrested form, or 25 of the 30 cases seen.

An epidemic occurred in May, 1910, in a rural school district in Hancock County, Iowa. Within a period of three weeks 30 cases of illness of the same general type occurred among 8 of the 12 families in attendance at this school; 5 cases resulting in typical definite paralysis were undoubtedly frank poliomyelitis. The remaining 25 may be considered, in all probability, abortive (arrested) attacks of the same infection. The most common symptoms in this group were severe headache, pains in the limbs and back, stiffness of neck and spine, and nausea and constipation. (Frost, Public Health Bulletin.)

Of 952 cases which occurred in Norway in 1905, 358 were of the abortive type. (Harbitz.)

It would seem that the closer the observation in any given epidemic, the greater are the number of cases of the arrested type described. In Wickmann's study of the epidemic in Sweden in 1905, he reported 157 arrested cases among 1025 cases he investigated,—15 per cent. He considered that the proportion of arrested cases was much greater than this, and was able to verify his belief in the smaller communities, where all cases could be more easily traced. He found:—

	Total.	Arrested cases.	Frank cases.	Per cent.
Trastena	49	23	26	46
Atvidaberg	31	11	20	35
Smedjeback	50	28	22	56

In the epidemic of 700 cases which swept the Island of Nauru in January, 1910, only 50 cases had any paralysis remaining at the end of three months, and Müller reported that many others had only a slight paresis lasting two weeks or less, while many had no paralysis whatever.

These cases are often unrecognized. I am reminded of the reply of a mother whom I saw in my daily round during

the Eau Claire epidemic. The mother was holding a lusty boy in her arms, and to the question if he was ill, said: "No, he ain't sick, he had a high fever last night, and this morning he can't stand alone, but he ain't sick." Those fleet symptoms marked the only disturbance of the 2-year-old.

The motor weakness of a small child in arms could appear only as an unnatural limpness. The arrested case may terminate fatally as in the one recorded:—

Case 7. Child 2 years old. One of twins. (Twin polioencephalitis with coma and paralysis of right upper and lower extremities.) Onset four days after onset in twin. Vomited, fever, stupor, some cervical rigidity, ataxia, no palsy. Temperature normal on eighth day. Child up at end of second week; case considered abortive and was about to be discharged when "she suddenly developed convulsions and died." (Sophian.)

Symptoms of the Arrested Type.—The symptoms of onset of poliomyelitis are the symptoms of the arrested form of the disease. These symptoms usually occur in a somewhat modified form, but they may be as severe and prolonged as a case of the paralytic type. Some symptoms are apt to be more prominent than others, and cases of the same type usually occur in groups. The arrested forms in a family which develops one or more cases of true paralysis, may all suffer from a neurasthenic hyperexcitability or languor; the members of such a group may all be attacked with nausea or vomiting, or diarrhea, or both. Meningism may mark the onset in another community. The symptoms of a general infection, some degree of gastric disturbance and meningism, are present in all these cases. Constipation with a preceding diarrhea is the rule.

A change in disposition is the most common symptom; basilar headache with cervical tension is usually present. There is a rise in temperature, which is usually brief, lim-

ited to a few hours; but it has lasted into the third day. The pulse rate is increased. There is a feeling of marked languor, or one of anxiety and restlessness. Anorexia is usual, and there may be nausea and vomiting. There is also some degree of pain. This may be a myalgic pain of the back or extremities, and is associated with tenderness.

Fig. 39.—Poliomyelitis involving the neck. Note forward "head-drop." (*Sheffield.*)

The spine is usually tender its entire extent. Tremor, in-co-ordination, an ataxia manifesting itself as an apparent clumsiness may or may not be noted. When a frank disturbance of motion is apparent, or meningitic twitchings or convulsions occur, the case does not belong to the arrested type.

Cases of the arrested type will be found out playing on the first, second, or third day, but walking with their heads

well back and a somewhat stiff and awkward gait; a leg may occasionally give way, causing the child to fall. (Anderson.)

The diagnosis of the arrested type of the disease is not difficult during an epidemic, when the association with frank cases is apparent. Without this association, but during the present pandemic, it would be well to regard with suspicion cases of basilar headache, with cervical tension and tender spines, which at the same time present an elevated temperature.

II. THE SPINAL MYELITIC TYPE.

Case (typical, mild). D. E., 3 years; male (physician's son). Onset: temperature 104° F.; twitching, but no convulsions; delirium; stupor; pain; paralysis of right leg; knee-jerk absent on the affected side, normal on left; slight atrophy; recovery. (Dr. A. W. Myers, Milwaukee, August 10, 1908.)

Case (typical, severe). D. H., female, 2 years. Onset: temperature 103° F.; pain; vomiting; delirium; paralysis of both lower extremities, left upper extremity, muscles of back and neck; remains same. (Dr. M. W. Dvorak, La Crosse, Sept. 16, 1908.)

The characteristic of the spinal myelitic type is the development of a flaccid motor paralysis of the muscles supplied by spinal nerves. The distribution of the paralysis of the muscles usually follows their segmental relation in the cord. The usual involvement of the lumbar enlargement and the less frequent destructive invasion of the cervical enlargement result in the paralysis of the lower and, less commonly, of the upper extremities. As a rule, the paralysis is more extensive than persistent, a regression taking place after the height of the paralysis has been attained. The severity of the onset is no certain indication of the degree of paralysis which may obtain.

The spinal myelitic type is most apt to present an aura of onset; this aura usually occurs from two days to a week

before the onset and will take the form of a stumbling gait, unusual falls in a hitherto sure-footed child, and tremor, inco-ordination, and ataxia in the adult.

The onset is sudden, with fever and general indisposition: insufficient observation is responsible for the statement that cases of paralysis occur without preceding initial

Fig. 40.—Paralysis of right leg, left arm, and face, with pain and hyperesthesia. (N. Y. Hospital for Deformities and Joint Diseases.)

symptoms. Sometimes the attack develops in two distinct periods with a pause between, during which the patient recovers, and then suffers a relapse and the paralysis declares its presence.

The paralysis usually develops from the second to the fifth day after the onset. It may be delayed for a longer period, two, three, four and six weeks' delay having been reported. It is progressive in development. If the lower extremities are attacked, paralysis of one develops twenty-

four to forty-eight hours before the other. The lower limbs are usually involved before the arms, although the reverse may occur. If the paralysis is not suspected, it may have fully developed before it is discovered; this is not infre-

Fig. 41.—Paralysis of muscles of left abdominal wall, with hernia. (N. Y. Hospital for Deformities and Joint Diseases.)

quently the case with young children, and gives rise to the statement that complete paralysis develops suddenly.

With the progressive development of the paralysis, the symptoms of onset diminish in intensity and usually by crisis. The greatly accelerated pulse slows to near the normal, or may show a bradycardia. The febrile temperature drops to within one degree of normal. The respira-

tions also become less frequent, unless there is an oncoming paralysis of the respiratory muscles.

The symptom groups which originate in meningeal irritation or in invasion of sensory fiber tract may show some remission, but usually become more profound until regression of the paralysis is established. Pain and tenderness of the spine are enhanced, and great hyperesthesia develops in the affected extremities. Delayed urination or retention occurs, due to paretic inhibition of the bladder wall; the paretic torpidity of the bowels results in coprostasis.

Considered as a whole, however, the symptoms of onset moderate to a marked degree or disappear with the oncoming of the paralysis.

Progression of the paralysis to its maximum limit and extent may occupy from two days to a week or more. There may be a paretic condition only, with quick return to normal function of the involved group of muscles; there may be paralysis of apparently severe degree involving all four extremities, and a subsequent recession of the paralysis, with no permanent damage to more than one extremity or muscle group. A recession of the paralysis is usual, but a certain number of cases show no regression, and a subsequent atrophy of the involved muscle groups occurs.

The muscle groups least involved and those last involved usually recover first; the regression may leave but one token of paralytic attack, the ptosis of an eyelid, or the involvement of a single muscle group, leaving a wryneck or drop-foot.

Distribution of Paralysis.—The distribution of the paralysis in the cases which occurred in the State of Massachusetts in 1910 has been tabulated by Drs. Lovett and Sheppard:—

DISTRIBUTION OF EARLY PARALYSIS.

	Cases.
One leg only	145
Both legs only	146
One arm only	44
Both arms only	12
One arm and leg, same side	50
One arm and leg, opposite sides	18
Both legs and one arm	32
Both arms and one leg	8
Both arms and both legs	51
Ataxia (transitory)	7
Back	79
Abdomen	38
Neck	13
Respiration	39
Deglutition	12
Intercostal	1
Face	7
Right face	31
Left face	24
Strabismus	2
	759

(Lovett and Sheppard, Bulletin Mass. Board of Health.)

The lower limbs are most often paralyzed, and both legs are usually affected during the progression of the paralysis; the residual paralysis is usually confined to one leg. In our series of cases paralysis of both legs was reported (49 cases) to be about twice as frequent as paralysis of one leg (26 cases). With definite paralysis of one leg, examination of its fellow will usually demonstrate a marked exaggeration of the knee-jerk, which would imply some degree of involvement of the quadriceps extensor at least.

The lower limbs are affected much more often than the arms. Frost considers this ratio to be as 2 to 1. In Wis-

consin it was 3 to 1 during the acute stage. A severe case of the spinal type may present a paralysis of both legs and a spastic condition of the trunk and arms, which are further immobilized by pain during the acute stage, but the residual paralysis in these cases frequently involves the legs

Fig. 42.—Most common type of spinal form of poliomyelitis. Paralysis, atrophy, and shortening of one lower extremity. (N. Y. Hospital for Deformities and Joint Diseases.)

only. The cases presenting residual paralysis of leg or arm only would give a ratio of 8 to 10 of the lower-segment group to 1 of the upper-segment group.

A paralysis of any or all extremities may occur, or of any combination of these four members of the body. This

paralysis is rarely total at the maximum; the toes or fingers can usually be voluntarily flexed. Regression of the paralysis usually leaves a permanent lesion of only certain muscle groups: the great quadriceps extensor and peroneal group of the legs; the shoulder and upper-arm group of the arms. The flexors of the leg are rarely involved, and ex-

Fig. 43.—Spinal type. Actual shortening one and one-half inches in two years. (N. Y. Hospital for Deformities and Joint Diseases.)

tensors of the arm usually escape. The atrophy which follows rapidly often brings to notice a hitherto unsuspected muscle involvement; the buttock muscles of the affected leg wither, and a quickly established scoliosis is found, due to atrophy of the hip, side and back, as well as to the effort of the child to stand straight on the affected leg.

In the clinic, drawn largely from the east side of Manhattan, there are numerous cases of scoliosis due to the paralysis of the erector spinæ segments, with paralysis and atrophy of the serrati and latissimus dorsi. The lesion is usually unilateral: in 24 of 25 such cases seen at the

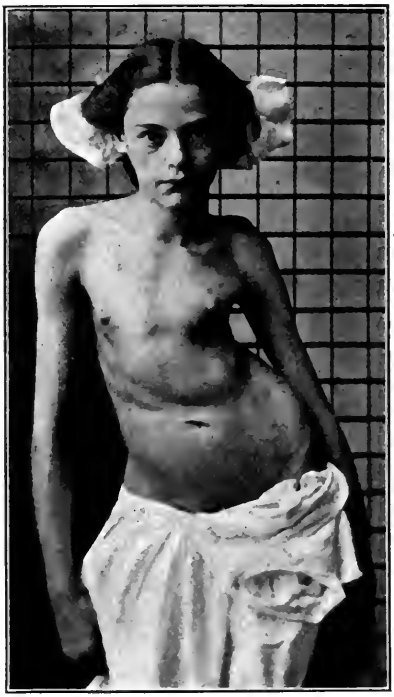


Fig. 44.—Paralysis, atrophy, scoliosis, and rotation following acute poliomyelitis. (N. Y. Hospital for Deformities and Joint Diseases.)

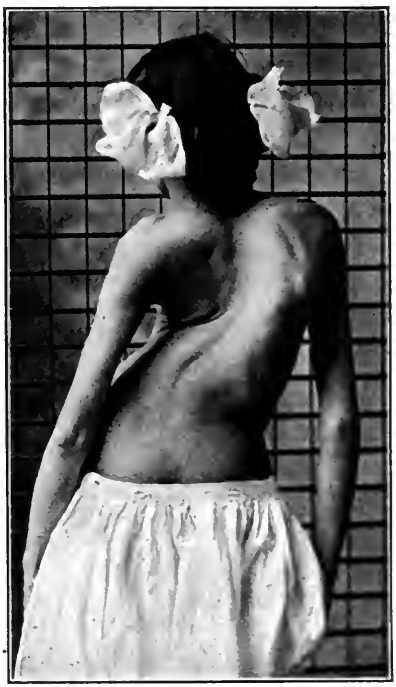


Fig. 45.—Posterior view of Fig. 44.

clinic of the Hospital for Deformities and Joint Diseases, the right side was affected.

The postparalytic atrophy of these muscles of the back and one side is extreme. The resulting distortions are various, depending largely on the contraction of the corresponding muscles of the opposite side.

Radiograms of these children show not only every degree of scoliosis, but a rotation of the bodies of vertebræ, from the pull of unopposed muscles.

In the Massachusetts epidemic of 1909, of which 613 cases were investigated, there was paralysis of the back, 83 times; of the abdomen, 37 times. In New York State, 1910, of 226 cases, the back was affected 34 times, the abdomen 20 times. No comment is made, however, in those cases of extreme distortion resulting. I am inclined to attribute the extreme cases seen at the New York City clinics to be the result of infantile paralysis, plus the conditions of tenement-house life.

Paralysis of the extremities is more common, but less serious than paralysis of the muscles of the torso. The gravity of the condition increases as the paralysis approaches the chest muscles. Some paresis of the involuntary muscles of the bowels is indicated by the constant constipation, to which is added, infrequently, a paralysis of the abdominal musculature. The meteorism attending paresis of the bowel pushes out the relaxed abdominal wall in huge hernias; three of these great protrusions are sometimes seen on a single case. These paralytic hernias are usually temporary, but may remain. When the paralysis is confined to one-half of the abdominal wall the unopposed muscles of the other side will draw the navel away from the hernia. The abdomen may be uniformly dome-shaped, with bulging during crying or coughing.

Paresis of the bladder is frequent, resulting in retention. Paresis of the urinary sphincter is less frequent, and incontinence may be the result of distention and overflow. Control of both urinary and anal sphincter is wholly lost, however, in a small number of cases.

Diaphragmatic paralysis is serious, but not of necessity fatal. It is indicated by a reversal of the abdominal respira-

tory excursion, which now retracts on inspiration, and protrudes the abdominal walls on expiration. When combined with paralysis of the intercostal muscles death from respiratory failure ensues. Paralysis may affect the muscles of one-half of the chest only, which will be completely immobilized, while the respiratory excursion may be seen on the unparalyzed side. When the intercostals of both sides are involved the chest will remain immobile, and breathing will be purely abdominal in type.

N. K., male, 11 years. Onset: fever 103° F., weakness, pain in shoulders, stiff neck; paralysis, two lower, two upper, diaphragm and throat muscles; recovery with some remaining paralysis of diaphragm. (Dr. Riley, Elk Mound, Wisconsin, September 6, 1908.)

TIME OF APPEARANCE OF PARALYSIS AFTER ONSET OF FEVER.

	Cases.
Same day	20
One day	31
Two days	40
Three days	34
Four days	15
Five days	11
Six days	11
Seven days	14
Eight days	4
Nine days	2
Ten days	2
Eleven days	2
Twelve days	4
Thirteen days	1
Fourteen days	1
Two to three weeks	5
Three to four weeks	1
Four to five weeks	1
Eight weeks	1
	200

(Lovett and Sheppard.)

Spontaneous Regression.—As a rule the paralysis is more extensive than persistent. When the paralysis reaches the maximum, regression is soon established in all but extremely serious or fatal cases. This improvement may be rapid for some days or weeks. The regeneration is not so rapid, but is continued *over a long period of time.* The spontaneous improvement in these cases is sometimes remarkable.

TRANSVERSE MYELITIS.

A variation in the usual spinal type of poliomyelitis is found when the involvement is distinctly segmental in localization and extends across a section of the dorsal or lumbar level invaded. Harbitz and Scheel demonstrated the pathologic lesion in a case of poliomyelitis which came to autopsy, and state: "We found the process affecting most the tenth and eleventh dorsal segments as a transverse myelitis with its symptoms, but with the usual diffusion of the process elsewhere."

Cases of transverse myelitic form of acute poliomyelitis have been reported by Williams, of Washington, and Skoog, of Missouri; an abstract of Dr. Skoog's very full report is included:—

F. G., schoolgirl, white, aged 13. Excellent health prior to date of onset; Nov. 2, 1909, pain in left lumbar and pleural region, following day along entire spine with backache, headache, legache, and pain in abdomen. These symptoms continued for four days with temperature to 103.2° F., respiration 24, pulse to 120. Constipation continuous, and retention of urine after third day. Patient wakened on third day by a sensation of numbness which began in both feet and crept rapidly upward to lower chest. The right and left sides were equally involved. A mild delirium was observed on the third, fourth, and fifth days.

Examination on the Sixth Day of Illness.—Mental depression slight, but coherent replies to all questions; normal function of cranial nerves; motor power in neck group and upper extremities

normal. There was an absolute flaccid paralysis of all muscles in the lower extremities, not a trace of movement being detected at hip-, knee-, ankle-, or toe- joints. The abdominal muscles showed much weakness. Complete retention of urine and feces. Reflexes of head and upper extremities normal. No epigastric, abdominal, gluteal, patellar, or ankle reflexes could be elicited. Plantar stimulation produced no response. There was a complete sensory paralysis of the area supplied by the eighth dorsal cord segment and below, there being no response to touch, cotton, pinpricks, thermal test, deep pressure or vibration stimuli in the lower extremities. Movements of the thorax involving all the upper dorsal vertebræ caused intense pain, but there was no true hyperalgesia nor hyperesthesia from the seventh dorsal area upward. Sense of position was entirely absent at toe, ankle- and knee- joints, but feebly present at hips, the right equal to the left. Course: exhaustion, cystitis, bed-sores, and other complications, with emaciation reaching a severe degree, and death February 22, 1910. Full mental faculties were retained until forty-eight hours before the exitus, when they dulled into coma. The muscles of the lower extremities remained functionless, flaccid, and became rapidly atrophic. A feeble right and left toe extension sign could be demonstrated in the third week, but not two weeks later. No change appeared in the findings as to deep reflexes. Trophic disturbances developed early and edema of the lower extremities during the last month. There was continuous pyrexia to 104° F. During the early period of the illness, with considerable caution, a diagnosis of acute epidemic poliomyelitis with a transverse myelitis was made. The subsequent course of the case and the autopsy supported this diagnosis.

CARDIAC PAIN AND CARDIAC INVOLVEMENT.

Potpeschnigg, of Stuttgart, states that in some instances sharp pains in the breast were among the first symptoms of onset noted. It may very well be the case that the nerves supplying the respiratory muscles, which later develop a fatal paralysis, exhibit an early neuritis. A second possible explanation is involvement of cardiac nerve-filaments; the alarming rise in the pulse rate, which is a very constant factor of the onset, indicates that there

is marked cardiac involvement. The following cases of endocarditis in previously healthy children occurring synchronously with acute poliomyelitis were personally communicated to the writer by Dr. Benj. Ayres, of Brooklyn:—

ENDOCARDITIS AND ACUTE POLIOMYELITIS, WITH SYNCHRONOUS OCCURRENCE.

Case 1. F. W., female, 11 years. Onset July 8, 1912. Gradual. Paralysis appearing July 12th. Maximum paralysis in right arm and leg. Residual paralysis slight, February 15, 1913. Atrophy improving under massage and electricity; history of exposure, unknown; insect bites, none.

Heart findings: endocarditis from onset, improved after three months' treatment. Murmur and slightly accelerated, irregular action at present.

Case 2. G. B., female, 9 years. Onset March 1, 1912; sudden in character; paralysis date after onset, 8 days; maximum extent, left leg and arm, muscles of neck, and cervical region; paralysis residual, none after six months; atrophy slight. History of exposure, unknown.

Heart findings: endocarditis, onset on eighth day. Murmur and accelerated, irregular pulse still persist. (Ayres.)

III. THE ACUTE ASCENDING OR RAPIDLY PROGRESSIVE FORM. (LANDRY'S.)

Those cases of acute poliomyelitis in which there is progressive involvement of the cord present characteristic symptoms which align them clinically in a separate grouping. The progression of the paralysis is usually from the lower extremities upward, and death ensues, when both diaphragm and intercostal muscles are involved, from paralysis of respiration. The cases usually succumb during the first week, and some cases are incredibly swift in termination. Of 3 fatal cases of the acute respiratory type seen by the writer, a girl 5 years of age, whose onset occurred

after some well-marked prodromes, at breakfast, died between 5 and 6 o'clock of the same day; 2 brothers, aged 2 and 7 years, taken ill on Tuesday and Thursday, died of respiratory failure the following Sunday afternoon.

A classical picture of this form of poliomyelitis is given by Draper, Peabody and Dochez in their recent monograph, a part of which is abstracted:—

"The typical clinical picture is that of one with a clear, alert sensorium, fighting for every breath until he is literally suffocated. In fatal cases there is usually a pause after the acute onset of the paralysis. There may be one or two days without any definite increase in paralysis, but it is noticeable that the children are not doing so well as those that will eventually recover. Often the respiration is more rapid and a trifle more difficult than the degree of paralysis warrants. They are frequently unusually excitable and irritable. Then the paralysis may begin to increase. A laryngeal disturbance with hoarseness, aphonia, or difficulty in swallowing may be the first evidence of the spreading lesion. If the intercostals are still active the movement of the chest becomes less marked. If the diaphragm has hitherto been intact, its movement, as represented by the abdominal wall, becomes weaker, or there is an asymmetric movement suggesting a paralysis of one side of the diaphragm. The alæ nasi dilate with inspiration, and the accessory muscles of respiration of the neck come into play. As the diaphragm weakens, the neck muscles become more and more prominent until it seems as if the whole work of breathing depended on them. The head is thrown back, and with every breath the lower jaw is pushed downward and forward in an attempt to get air. Meanwhile the lungs may have remained perfectly clear until the very end, or a few hours before death coarse, moist râles may accumulate, an edema suggesting vasomotor paralysis. Heart sounds have been audible for as much as five minutes after breathing stopped. Several times a characteristic arrhythmia has set in for the last few hours of life. It is interesting that in one case, in association with the institution of artificial respiration and a lessening of cyanosis, the irregularity of heart action completely disappeared.

"With the onset of respiratory difficulty, it seems as if the children were suddenly awakened and made to realize the struggle

before them. One sees a sleepy baby become all at once wide awake, high strung, alert to the matter in hand, and this is, breathing. The whole mind and body appeared to be concentrated on respiration. The child gives the impression of one who has a fight on his hands and knows perfectly well how to manage it. Instinctively he husbands his strength, refuses food, and speaks when necessary with few words. One little child, aged 4, unable to move, but with a mind that seemed to take in the whole situation, said abruptly to the nurse, between her hard-taken breaths, 'Turn me over.' 'Scratch my nostril;' and then, to the doctor, 'Let me alone, doctor.' 'Don't touch my chest.'

"Pressure on the chest, tight neck-bands, anything that obstructs easy respiration is immediately resented. The child is nervous, fearful, and dreads being left alone. He often shows an instinctive appreciation for the specially efficient nurse. The mouth becomes filled with frothy saliva which the child is unable to swallow, so he collects it between his lips and waits for the nurse to wipe it away. The pallor is distinctive, the lips blue, cyanosis absent, and sweating profuse. The mind becomes dull, unconsciousness follows, and an hour or more later respiration ceases."

As a typical case may be considered the following:—

Werner A., 14 years. For three days, fever and severe constipation. On third day, paralysis of both legs, next day ascending to abdominal and thoracic muscles. On the fifth day, death from respiratory failure. (Kelly, Seattle.)

Case of Ascending Type, with Death on Fortieth Day.—E. F., male, 18 months. Onset, May 26, 1908. Fever, restlessness, delirium; pain on moving limbs; temperature when first seen (after paralysis began) 100.5° F., late, thirty-eighth day, 108.5° to 110° F., per rectum. Rash: bright first two days. Paralysis of both lower extremities followed by partial paralysis of both arms; inability to lift head; left facial paralysis and convergent strabismus of left eye. Patellar reflexes abolished; Babinski's sign present; twelfth day, motion returned to arms; fourteenth day, motion returned to limbs; fifteenth day, paralysis of rectus subsided; eighteenth day, involvement of hypoglossal; twenty-fourth day, twitching of arms and legs; twenty-sixth day, spastic contraction of hands; thirty-seventh day, crowing spasms of larynx; thirty-eighth day, tempera-

ture 108.2° F., per rectum; respiration rapid and irregular, becoming Cheyne-Stokes in type; fortieth day, moist râles over lower lobes of lungs, and dullness over same area. Death occurred on fortieth day, at 5.30 P.M., the temperature (rectal) one hour previous being 110° F. and pulse 154. Dr. Fellman's diagnosis was "infantile paralysis of cerebral origin." (Dr. G. H. Fellman, Milwaukee, Wis.)

Fig. 46.—Spinal type. Paralysis of serrati, latissimus dorsi, and erectors of spine: atrophy; scoliosis. Anterior surface. (N. Y. Hospital for Deformities and Joint Diseases.)

Fig. 47.—Same as Fig. 46. Posterior surface.

The paralysis in this case was of the ascending type; we are indebted to a careful observer for this first record of great disturbance of the heat center as shown by a rectal temperature of 108° to 110° F. It probably occurs and is overlooked in many cases of poliomyelitis. The steady progression of the paralysis, which in this case was much

prolonged, is shown by the late involvement of the cortex (spastic contractions twenty-sixth day) and final involvement of centers of respiration and heat.

Case of Descending Type with Death on the Fourth Day.—V. H., female, 3 years and 9 months. August 27th, vomited pear; 28, vomited everything given her, including water; played about, was sleepy; 8 P.M., physician called; temperature 102° F.; knee-jerk present, pupils reacted; Kernig absent. Gave calomel and ordered baths and ice to suck; 29th, temperature 100.3° F.; vomited everything given her; slightly jerky; no other symptoms. 3.30 P.M., all attempts to drink choked her and liquids came out through the nostrils; with some ejection of frothy substance from mouth; 8 P.M., throat filled with saliva and air-bubbles, regurgitated one-half teaspoonful of water; 9.30 P.M., temperature 100.5° F.; pulse good; child looked well except for paralysis of deglutition. August 30th, 2 A.M., child dying; no convulsions; no other paralysis; died at 4.30 A.M. (Armstrong and Cowern, 17 cases of poliomyelitis at St. Paul, Minn., in 1909.)

The separation of the acute ascending (or descending) paralysis from the spinal type is wholly arbitrary, and according to Wickmann such cases belong to the spinal type unless there is a fatal involvement of the muscles of respiration.

The paralysis in these cases makes steady progress, upward or downward from the area first involved, until paralysis of respiration closes the scene.

IV. ACUTE BULBAR-PONTINE TYPE OF POLIOMYELITIS.

Case of Bulbar Type with Moderate Cortical Involvement.—The patient, a child of 2½ years, could not protrude the tongue; not only was the left side of the face paralyzed, but there was oculomotor paralysis and motor paralysis of the fifth nerve, with the consequent strabismus and ptosis, and inability to close the jaws. This was later associated with a spastic paralysis of the right arm and leg, showing a spread of the lesion and the involvement of the upper motor neurons to the limbs of the opposite side

of the body. The child eventually made a good recovery. (Dr. Colin K. Russel, Montreal, 38 recent cases; a study of poliomyelitis.)

Case of Bulbar Type with Cranial-nerve Involvement Only. —W. C., male, 6 years; fever, headache, stiff neck, constipation; unconscious for several days; difficult breathing; inability to swallow; loss of hearing and speech for eight days; eighth day, right facial paralysis; difficult mastication. Facial and hypoglossal paralysis lasted for six weeks. Complete recovery. (Dr. Kelly, Bulletin Washington State Board of Health.)

In the bulbar-pontine form there is paralysis of the muscles supplied by nerves which take their origin in the medulla or pons; the cranial nerves most often involved are the facial, hypoglossal, and ocular. There may be involvement of the throat and larynx. Cases of the bulbar-pontine type may be associated with a spastic paralysis due to cortical involvement, as in the first case above given, with a lower segment paralysis; with acute respiratory paralysis and death due to invasion of the vital centers which are disposed along the floor of the fourth ventricle; or to tremor and ataxia due to interruption of the conducting fibers from the cerebellum.

Facial paralysis is the most frequent manifestation of this type; it is usually unilateral, but may be bilateral. It is frequently the only manifestation of the acute disease, and in sporadic form occurs not infrequently among adults. A considerable number of cases among adults are constantly in attendance at the clinic of the New York Hospital for Deformities and Joint Diseases.

Five per cent. of the cases in the Massachusetts epidemic of 1909 had facial paralysis.

Ocular disturbances are common. There may be a transient nystagmus or diplopia. Internal squint, due to involvement of the external rectus, and divergent squint with ptosis, from paralysis of the oculomotor, are often seen. There may be fixation from paralysis of all the

muscles. There may be transient blindness; or optic atrophy with permanent blindness.

Stephenson, of London, has observed a group of cases in children the chief characteristic of which is the sudden onset of strabismus at an age when ordinary squint is not altogether common. The child, often apparently in usual health, is put to bed, and on awakening next morning is found to present squint. Stephenson's observations lead him to conclude that the oculomotor symptoms in these cases depend on an acute focal encephalitis, quite analogous with the better-known forms of that disease. He has seen 28 such cases, all the patients being under 6 years of age, and half of the number under 1 year. Although the paralysis may affect any of the extrinsic muscles of the eyeball, yet in three-fourths of the cases the external rectus muscle alone is involved. The extrinsic musculature of the eye is seldom attacked. Stephenson says that the common form of encephalic strabismus is very apt to be confused with ordinary concomitant convergent strabismus.

Transient aphasias and transient deafness are not rarely seen in this type of poliomyelitis. Dysphagia, with salivation and regurgitation of all liquids through the nares, is frequent. Dyspnea and the Cheyne-Stokes syndrome, when there is no paralysis of the chest muscles, point to alarming involvement of the pneumogastric centers.

V. ENCEPHALIC TYPE.

Cases of spastic hemiplegia with resultant contractions, but no atrophy, are frequently seen in close association with cases of the flaccid paralytic type. The association may occur in two members of the same family, and this association is not infrequently seen in one individual who presents, after the acute attack, both spastic and flaccid lesions.

Fig. 48.—Atrophy and lordosis with rotation following upper segment paralysis, with paralysis of serrati, latissimus dorsi, and erector spinæ. Anterior view. (N. Y. Hospital for Deformities and Joint Diseases.)

Fig. 49.—Same as Fig. 48. Posterior view.

Spastic lesions arise from injury to the motor cortex of the cerebrum, or destructive invasion of its paths of conduction.

A spastic paralysis, however, is but one of the results of an encephalitis produced by the virus of poliomyelitis.

There may be associated with the spastic paralysis: tremor, an acute ataxia, athetosis, and clouded mentality.

The association of a flaccid paralysis of the extensors of one or both legs, with a spastic condition of the great

Fig. 50.—Oculomotor type. Strabismus. (N. Y. Hospital for Deformities and Joint Diseases.)

toe or toes, or of the fingers of the hand of the opposite side, is seen so frequently in the clinic of the New York Hospital for Deformities as not to arouse comment.

Three cases, the first and third of which were seen at this clinic, are given:—

Hospital for Deformities, Examining Room, May, 1911. M. K., 4-year-old girl, of Irish-American parentage; well developed; conscious; carried in by mother. Onset ten days previous, with fever and vomiting. Child spastic and rigid from head to heels; spasticity increased on handling; when placed on feet on examining table child was rigid as a bottle and could be passed back and forth between the hands as a bottle might be if tapped lightly on the neck;

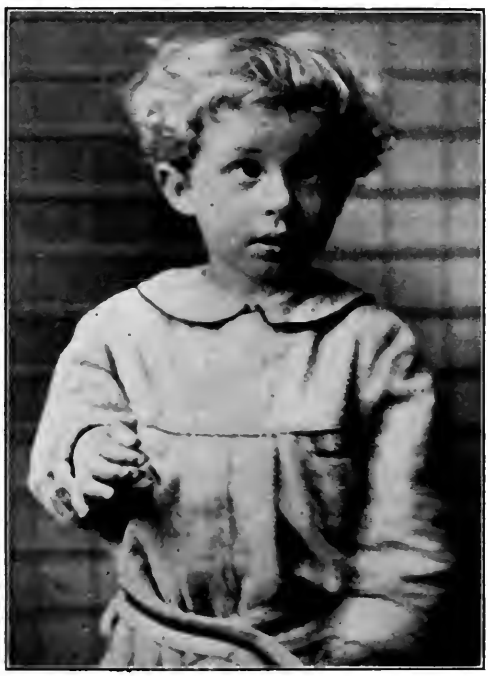


Fig. 51.—Oculomotor type. Strabismus. (N. Y. Hospital for Deformities and Joint Diseases.)

this action increased the spasticity until the child was standing rigidly and involuntarily on tiptoes.

Armstrong and Cowern, of St. Paul, reported 17 cases of poliomyelitis, in 1909.

C. J., aged 6 years, female; Sept. 3d, malaise and headache; Sept. 4th, feverish, drowsy, constipated; Sept. 5th, 5 P.M., first seen by physician, who considered it a case of indigestion, gave calomel,

and ordered citrate of potassium, which was vomited. Vomited several times; told her mother her left hand hurt her and "wanted to stay shut." Sept. 6th, paralysis of hand; could not extend fingers. Child was up and appeared well save for a "wobbly" gait. Feb. 19, 1910, child carries left hand in right; with effort she can extend fingers and thumb, and the fingers are in a state of semiflexion with distal joint of thumb semiflexed. This was a case of direct infection from a cousin.

Hospital for Deformities, May, 1911.—F. B., male aged 19 months. Well-developed boy; American parents; walked and talked at 14 months. Acute onset, April 19, 1911 (a sister also contracted the disease, but made a good recovery). High fever; head sweating; strabismus; opisthotonus; unconscious nine days. Five weeks later, paralysis of extensors of both legs; spastic right and left great toes; fingers of both hands spastic, and hands and arms constantly employed in slow, athetoid movement when awake. Constant slow, vermicular motion of torso; makes no effort to sit, stand, or talk.

Head hydrocephalic, circumference 18¼ inches; fontanels unclosed; mentality clouded, but recognizes parents; marked irritability.

A case of spastic paralysis in an adult has been reported by Drs. Anderson and Frost:—

Mrs. W., 22, waitress, was taken sick the latter part of June, 1910, with fever and indefinite general symptoms. After several days she became paralyzed in both lower limbs. She was admitted to a hospital about one week after onset. She was said to have had, at that time, a flaccid motor paralysis of both lower extremities, which, however, became spastic within a few days. When the patient was seen, the latter part of July, 1910, *both legs and thighs were quite spastic.* No active *motion was possible except of the toes and slight flexion of the left knee.* Passive motion was limited to partial flexion of the thighs and slight flexion of left knee—very little of the right knee. The patellar reflexes greatly exaggerated on both sides; ankle-clonus on right side; sensation for touch and pain was normal. Examination was otherwise negative. The patient's general health was good.

November 25, 1910, the left leg could be moved, but rather awkwardly. The right leg showed little, if any, improvement. Patellar reflexes were still exaggerated, more so on the right side. There was no ankle-clonus and no atrophy.

"The *spasticity of the paralyzed limbs,* exaggeration of reflexes, and *absence of atrophy* in this case indicated a lesion in the *upper motor segment, either in the motor cortex of the brain or in the pyramidal tracts of the cord.* The case was included in our series in order to ascertain the diagnosis, *since it represents a rare clinical type of poliomyelitis, the diagnosis of which has always been uncertain, and whose occurrence has been a matter of some dispute.*" (Anderson and Frost.)

In Sophian's series of 20 cases seen in a period of three weeks during the New York epidemic of 1911, 12 of the cases were of the encephalic type, that is, 60 per cent. of a group of cases of poliomyelitis presented a preponderating cerebral involvement. This is a possible result of epidemic poliomyelitis that is little known, and of extreme importance, and Dr. Sophian's brief table of the 20 cases is therefore given:—

TWENTY CASES FROM FALL EPIDEMIC OF 1911.

1. Myelitic form ..... 3 cases.
   All showed paralysis of both lower extremities.
2. Landry's ascending ..... 1 case.
   All 4 extremities, intercostals, and face.
3. Abortive form (one ataxic) ..... 4 cases.
   Slight, temporary quadriceps, paralysis with ataxia, 1 case.
   Temporary paralysis right side of face and arm, 1 case.
4. Polioencephalitic, cerebral forms ..... 12 cases.
   Hemiplegia in 5 cases; bulbar involvement in 3.
   Ophthalmoplegia (complete), 2 cases, associated with left facial palsy in 1 case, and with palsy of the left lower extremity in the other.
   Bulbar involvement, alone, 1 case.
   Bulbar involvement, with paralysis of one upper extremity, 1 case.
   (See 3 cases of bulbar involvement with hemiplegia.)
   Isolated paralysis, 2 cases.
   Left facial, right facial and right upper extremity, 1 case.

Left upper extremity, left side of face, internal left eye, 1 case.

NOTE.—*1 case* with left facial palsy, and a left-arm paralysis, the latter possibly myelitic in origin.

Ages of cases ranged from 3 weeks to 22 years. (Sophian, New York.)

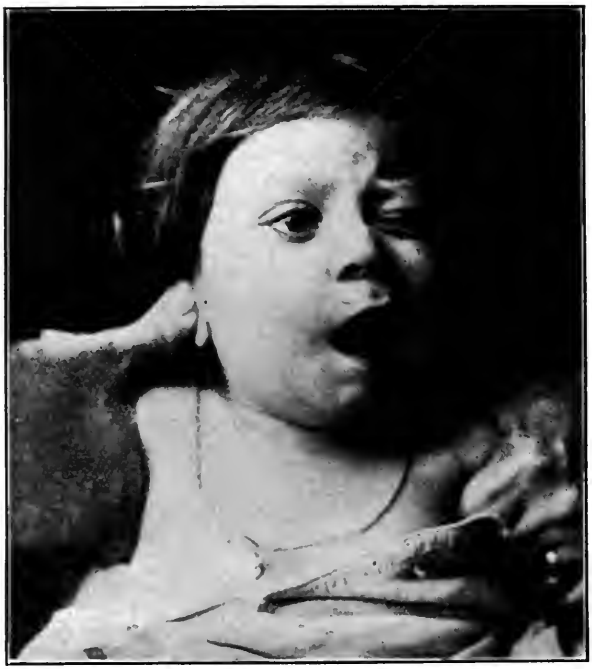


Fig. 52.—Acute bulbar type. Left facial. (N. Y. Hospital for Deformities and Joint Diseases.)

Classification of Symptoms of Encephalic Type.—Modified from Reginald Miller.

Polioencephalitis superior. Rolandic cortex-spastic hemiplegias (Strumpell's paralysis).

Frontal area—associated with mental defectives and morons.

Occipital area—blindness with normal eye-grounds and active pupils.

Symptoms common to all: Stupor, coma, meningitic cry, bulging fontanels.

Polioencephalitis inferior (bulbar-pontine type—see above). Paralysis facial, oculomotor—auditory—one side only. Tremor (pontine); bulbar paralysis (vital center of medulla).

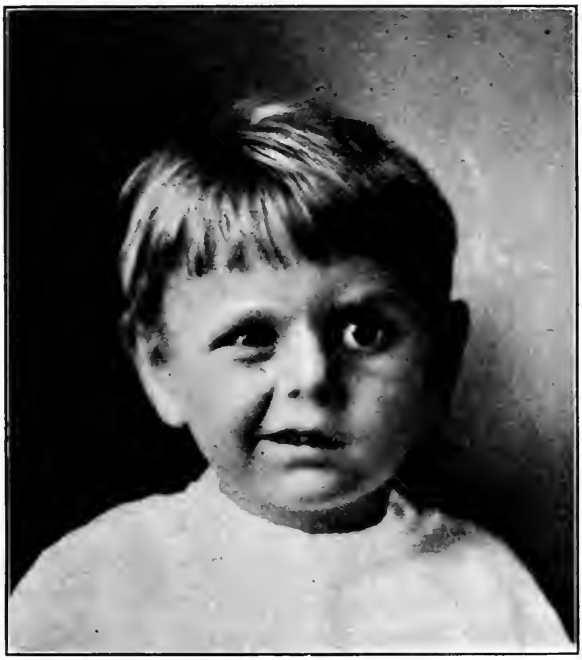


Fig. 53.—Acute bulbar type. Right facial. (N. Y. Hospital for Deformities and Joint Diseases.)

Encephalitis cerebelli (predominant ataxia type). Ataxia well marked or extreme; not demonstrable while patient is stuporous, evident when patient rallies and makes voluntary movement; nystagmus; scanning speech. (Clinical diagnosis confirmed twice: *post mortem* in 1 recent case and in 1 case of thirty years' standing.)

Encephalitis of midbrain and connections: Acute tremor; hypertonus; excessive emotionalism. Tremor, due

to the alternate action of groups of muscles and their antagonists; a slow, rhythmic movement of the intention type, at the rate of about five a second. It is of the intention type, and appears only when an attempt is made to use the affected limb. Hypertonus, not a true spastic condition, but sufficient to make the movement of limbs slow, stiff, and awkward.

Thalamic encephalitis: Spastic paraplegias and hemiplegias may have an associated athetosis or chorea due to lesions in the optic thalami. (See III, encephalic type, below.)

All types of encephalitis enumerated above may occur:—

I. In epidemic form.

II. In sporadic form.

III. As congenital cases from intra-uterine infection. Congenital spastic paraplegias. Mental deficiencies of all degrees.

Polioencephalitis of a pure type, with no paralysis, spastic or otherwise, may occur. This class of case among male adults is almost uniformly fatal, and is rarely recognized in its relation to the epidemic disease. Such a case, confirmed by the post-mortem examination, is here given:—

Anatomic Investigation of 19 Cases of Epidemic Acute Poliomyelitis.—Male, 39 years; fever; headache, stiff neck, vomiting, some rigidity of limbs, convulsive seizures, clouded consciousness; coma, death on twelfth day; no paralysis nor paresis.

Necropsy.—Diffuse hyperemia of central nervous system; softened encephalitic foci in the right temporal lobe and gyrus fornicatus of both sides. Inflammation extended with lessened intensity to basal ganglia, along aqueduct of Sylvius, through medulla oblongata and was even demonstrable in upper portions of cord. (Harbitz and Scheel.)

Gregor and Hopper reported 132 cases of poliomyelitis in Cornwall and Devon, England, in 1911. One case follows:—

O. N. B., male; onset August 28th, temperature 100° to 101° F.; vomiting. August 30th, very irritable. Kernig present on both sides; fundi normal; paralysis of external recti; no other paralysis. September 2d, semicomatose, gradually deepening to stupor. September 3d, coma, died.

Frontal-area Involvement, with Resulting Mental Defect.—Feeble-minded conditions subsequent to and caused by acute poliomyeloencephalitis have received little consideration as yet by investigators of the recent pandemics of the disease; the writer considers it probable that a majority of all morons, idiots, and imbeciles are victims of this acute infection of the nervous system. Of the few brief references in the literature there is one of Dr. Mark Crain, of Vermont, to the effect that he observed a child which became feeble-minded after an attack of the disease.

PREDOMINANT ACUTE ATAXIA TYPE.

In the foregoing classification of cases of polioencephalitis according to their cerebral localization, it will be seen that the cases of acute ataxia are included in the subhead "encephalitis cerebelli." While it is true that some degree of ataxia may be present at the onset of any case, there is a type of case in which ataxia of an acute and extreme degree has been the predominant feature. These cases are not common; and as it has now been demonstrated that they have their origin in a cerebellar or pontine crossing lesion, there is no necessity of giving them a further classification than their proper alignment in the encephalic group.

Acute ataxia is the prominent symptom of this group, developing suddenly and associated with systemic disturbance. While the patient is in a comatose state the ataxia will be masked, but becomes apparent as the patient convalesces. A child may attempt to stand by placing its feet wide apart, and to walk by taking short steps, but will

sway and fall. These cases will stand and step more readily when placed in a water bath hip-deep, but must be supported and watched while in the water.

Fig. 54.—Congenital spastic paralysis. (N. Y. Hospital for Deformities and Joint Diseases.)

The ataxia may be well marked and has been called a "wild ataxia" by Leonard Parsons. There can usually be found reflex extensor plantar responses. Nystagmus,

scanning speech, and hypertonus of muscles may be associated.

This acute ataxia was first described by Leyden in

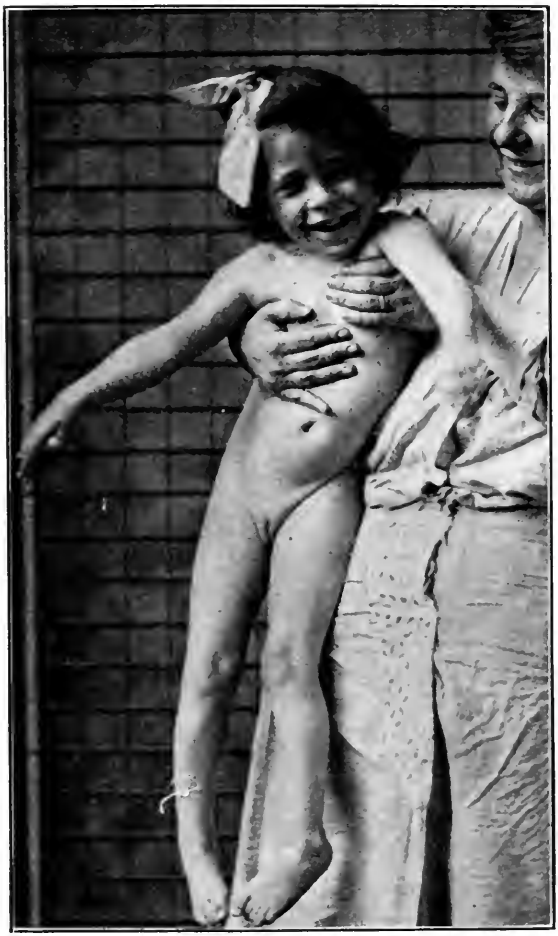


Fig. 55.—Same case as Fig. 54.

1891; a number of cases have been reported in England (Batten). The clinical diagnosis has twice been confirmed by autopsy. An early case was reported from Germany in

1895; a case of thirty years' standing was described by Clapton (Reginal Miller).

E. S. White, female, Kentuckian, 12 years; onset July 5, 1911, headache, fever, rapid pulse; movements impaired and locomotion embarrassed; August 18th, referred to Ohio Medical College Clinic.

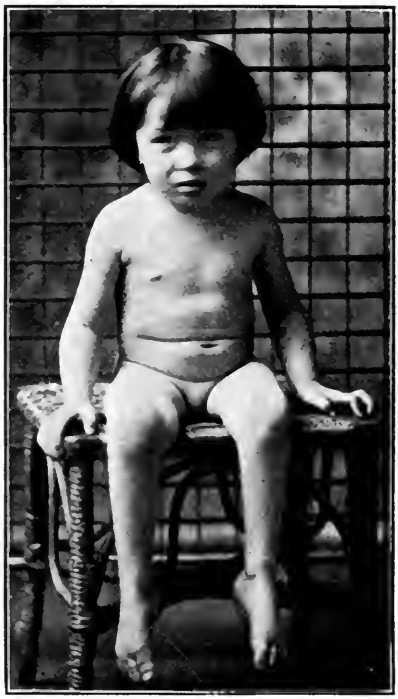


Fig. 56.—Bilateral drop-foot. (N. Y. Hospital for Deformities and Joint Diseases.)

Emaciated, weak, headache, projectile vomiting. Slight internal strabismus of left eye; right side of face paralyzed, tongue protruded to right, speech inhibited; sternocleidomastoid of right side a flaccid paralysis with peculiar position of head. A distinct cerebrospasmodic gait. A provisional diagnosis of brain tumor or basilar meningitis was made, when younger sister developed symptoms of infantile paralysis, with paralysis of both hips and later the father and another sister became ill. The cerebrospasmodic ataxia

of the first case persists. (Batte, Lancet-Clinic, Cincinnati, December 2, 1911.)

It is difficult and perhaps unnecessary to draw a sharp line between inco-ordination and ataxia. Both may precede the acute onset of poliomyelitis.

Fig. 57.—Shortening. (N. Y. Hospital for Deformities and Joint Diseases.)

Frost states that inco-ordination may be ascribed to several possible causes:—

1. Lesions of the cerebellum.

2. Lesions in the conducting tracts leading from the cerebellum.

3. Lesions of the posterior cornu of the cord, affecting muscle sense.

4. Paresis of certain groups of muscles, disturbing the

balance between these and their opposing (unaffected) muscles.

5. Peripheral neuritis.

To which might be added:—

6. Imperfectly developed conduction paths in the child, which are subject to disorganization from numerous causes.

As has been stated, the acute ataxia of predominant type arises from the first or second of these lesions, *i.e.*, of the cerebellum or its conducting tracts. It is associated with exaggerated (especially plantar) reflexes, transient coma, nystagmus, scanning speech, and usually terminates in recovery.

Three ataxia cases were seen by Sophian in three weeks, in the summer of 1911, among 20 cases studied during that period.

VI. MENINGITIC TYPE.

Case: Meningeal Type; Mild.—E. T., male, 8 years; Sept. 25, 1908; headache; pain in neck; severe pain in back; temperature 102° F.; no other case in house, but several in village. Comatose for several days; no paralysis; "meningeal type, no spinal symptoms." (Dr. Bowles, Eleva, Wis.)

Severe Case of Meningeal Type.—One case gave as exact a picture of meningitis as can be imagined. The child was unconscious, back bowed, buttocks and head sustaining weight of child if placed on back, spinal column as stiff as a board, spastic paralysis of arms and legs, rotation of eyeballs, mumbling and groaning, arms crossed rigidly over chest, and legs flexed and stiff from contraction of hamstring muscles. Temperature 104.2° F.; pulse 140; respiration very fast and labored. Such a case required close work to differentiate. A lumbar puncture gave a clear fluid which escaped under great tension at first. The specimen centrifuged and stained showed only mononuclear lymphocytes. There were no intracellular organisms and no polymorphonuclear cells. (Hallett and Shidler; Shidler, Pediatrics, 1910.)

It is the appearance of this confusing meningeal type of poliomyelitis early in the epidemic which has so often led physicians to consider that they were facing an epidemic of cerebrospinal meningitis. The meningeal symptoms are characteristic and may predominate. A paralysis

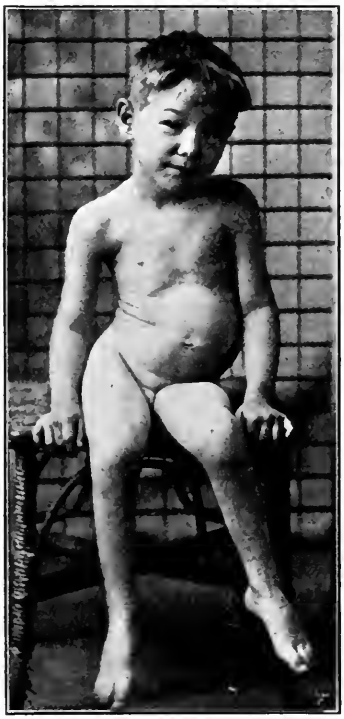


Fig. 58.—Scoliosis, drop-foot, and shortening. (N. Y. Hospital for Deformities and Joint Diseases.)

Fig. 59.—Posterior view of Fig. 58.

may follow which at once clears the diagnosis, but a certain percentage of the cases recover or die with no paralysis. Wickmann, in his great monograph, states that clinically and by autopsy it was demonstrated that the whole course in some of these cases was that of a meningitis serosa; he would also include sporadic cases of meningitis serosa as

due to poliomyelitis. The two cases given above illustrate a mild and very severe phase of this type of poliomyelitis. They conform absolutely to the classic conception of meningeal irritation: severe headache, pain in the neck and spine, retraction of the head, contractions of the spinal muscles, spasticity, and disturbed vision.

In the Vienna epidemic of 1909, Spieler observed 8 meningeal forms in a series of 44 cases; in 4 of these the picture resembled tuberculous meningitis so closely as to be diagnosed only late in the disease. The cases very frequently have a comparatively lengthy prodromal period, with beginning change in mood, fatigue, nightly unrest with frequent outcries, occasional vomiting with obstipation, suggestive of a beginning basilar meningitis. Then, in children, convulsions suddenly occur, marked stiffness of the neck, irregular pulse, increasing patellar reflexes which usually diminish later, facial pareses, strabismus, general hyperesthesia, and vasomotor disturbances. At this period the lumbar puncture will show lymphocytosis, but no purulent exudate. In the favorable cases the symptoms begin to recede, the temperature falls to normal, the irritative phenomena diminish, and there may develop a number of the palsies characteristic of the spinal form.

A case of the meningeal type occurring in an adult of 29 years has been reported by Dr. Jelliffe. (Jour. Amer. Med. Assoc., June 24, 1911.)

Poliomyelitis in pregnancy may take the meningitic form and simulate eclampsia with the utmost fidelity; such a case was reported by Wickmann in a young woman, six months pregnant, taken acutely ill, with retracted head and spine followed by severe convulsions. Forced delivery was successfully conducted; the convulsions continued, although the patient was entirely conscious until

death on the third day, when autopsy revealed typical histologic lesions of acute poliomyelitis.

With the meningitic symptoms enumerated, there may be paralysis; the patient may be wholly conscious, or suffer any degree of convulsive attack, stupor, delirium or coma. The only certain means of diagnosis is by lumbar puncture and examination of the spinal fluid.

VII. NEURAL TYPE.

In the neural type of poliomyelitis the onset is similar, but the pain is not confined to the basilar and spinal areas. Pain in the extremities assumes an agonizing character; there is tenderness on pressure along the nerve-trunks, and violent pains along the course of the nerves. The pain and tenderness is much more severe than is usually recognized, but the proximal areas of nerve-trunks will be found to be most affected, due to the acute process which is going forward in the spinal ganglia, while in multiple neuritis the distal areas of the periphery are exquisitely tortured. Wickmann says of this polyneuritic type that during epidemics cases occur which would be considered acute neuritis if they made their appearance as isolated cases. Clinically and etiologically they are cases of acute poliomyelitis.

Dr. C. A. Anderson states that 22 of 86 cases of poliomyelitis were of markedly neuritic type, with severe, intermittent, sharp pains in one or more members during the third day and after. The severity of the pain was usually in proportion to the extent of the involvement. The pains last from one-half to ten minutes.

In some of the cases a flaccid paralysis follows.

Cases of purely neural type are seen; the pain may be very persistent, and the case a long one of many weeks. Such cases present no paralysis, and the diagnosis is very confusing unless the relationship to a case of poliomyelitis

is evident. This form may be seen in adults or children, perhaps more frequently among adult women. Neuritis is such a rare disease in childhood that when it occurs infantile paralysis must be suspected, the neuritis of diphtheria having been previously ruled out.

The purely neural type is comparatively rare; pain of a multiple neuritic character may be observed in combination with any of the foregoing types of the disease. It differs from multiple neuritis in this particular: the tenderness is somewhat more extreme in the proximal areas of the nerve-trunks, while in multiple neuritis the pressure pain increases toward the periphery.

The pain, of a severe and intermittent character, is most marked along the course of the great nerve-trunks. It recurs at regular intervals, is increased by handling of the extremities, or movement of the patient.

The severity of the pain seems to be in proportion to the extent of the involvement. Paresthesias are frequent, and there may be a marked alteration of temperature sense and tactile conduction. The pain may be severe and intractable; it may disappear promptly in cases of the arrested type; it may become chronic and old cases of poliomyelitis complain of an ever-present sciatica.

The peripheral pain and soreness is supposed to be due to central involvement. No involvement of peripheral nerve-filaments has been found *post mortem* in poliomyelitis. Spontaneous pain and pain on pressure are pronounced; there may even be *objective sensory disturbances* in rare cases, and especially of the sensations of pain and temperature, from implication in the posterior horns of the fibers concerned with these sensations. While we recognize clinically *the neuritic form,* we have no pathologic evidence justifying this recognition, as no changes in the nerves have been noted. I believe, however, that this

failure of observation is not from failure of neuritic changes, but *from inability to study the nerves in cases in which neuritic symptoms have been pronounced.* Strauss reports an infiltration of the posterior-root fibers and the arachnoid covering of the spinal ganglia only. The association of herpes zoster with poliomyelitis would indicate that there may be an unrecognized form of peripheral neuritic poliomyelitis, or that herpes zoster is an expression of poliomyelitis, attacking the elderly and undernourished.

Poliomyelitis Affecting the Superficial and Deep Muscles of the Neck and Extremities; Resembling Polyneuritis.—The little girl was 3 years old, delicate from birth and subject to frequent "colds and coughs," apparently due to enlarged tonsils and adenoids. She had not been feeling well for two days; as the parents put it, "was terribly weak, had pain in swallowing, and refused to take any nourishment." As I went about to examine her she cried bitterly with pain. On one occasion a mouthful of water came back through the nose. The throat was free from a deposit, but deglutition was difficult. Her temperature was only 100° F. She was able to move her legs from one part of the bed to another, but complained of pain while doing it. The same was true of the upper extremities, especially of the hands. She could rotate her head laterally and there was neither retraction nor stiffness of the neck. We thus had: symmetrical paresis of the extremities, intense pain, difficult deglutition, regurgitation of fluids through the nose and nasal tone of voice (which she always had owing to the adenoids)—*i.e.*, a typical picture of post-diphtheritic neuritis—and yet no history of diphtheria or of any other grave disease within six weeks previous to this attack. I ordered 3 grains of sodium salicylate and $\frac{1}{100}$ grain of strychnine to be given every three hours.

The next day she was entirely free from pain and apparently happy.

No longer objecting to the examination, I went over the case most carefully, and as I propped the child up against the pillow to give a real good look at her throat I was surprised to note that her head dropped forward upon the sternum like a dead weight, the patient being unable to lift it in position. On removing the pillow and placing the patient in slanting position, the head dropped also forcibly backward. She has gradually greatly improved, though she still (four years after the attack) shows lack of strength in the anteroposterior mobility of the head. With all our latest scientific data on the subject, I am not aware of any diagnostic means which would have enabled to differentiate this case from polyneuritis at so early a stage. (Sheffield, New York.)

Dr. S. Leopold, of Philadelphia, writes: "I wish to call attention to a case seen with Dr. Pittfield which is rather interesting, because it was one of the neuritic type of poliomyelitis of Wickmann. This case is the only one on record, so far as I am aware, in which the opportunity occurred of studying the peripheral nerves. The case presented the ordinary type of poliomyelitis with acute onset, paralysis of the lower limbs, which, however, was unsymmetrical, and tenderness over the nerve-trunks, during the entire period of illness, lasting two months; the knee-jerks were lost, and there was bladder disturbance. The case had been diagnosed as multiple neuritis.

"At necropsy I found a true picture of the reparative stage of poliomyelitis, but I found the curious fact that the peripheral nerve, which was examined and placed in osmic acid immediately at necropsy, showed practically no degeneration that could account for the tenderness, while the spinal cord showed an intense meningitis; most significant

was the involvement of the posterior roots, the exudate being in the posterior root. The congestion of the vessels was intense; and I believe that the tenderness over the nerve-trunks was of central origin. The case is rather unique in the history of poliomyelitis." (Jour. Amer. Med. Assoc., Sept. 28, 1912.)

Herpes Zoster.—Herpes zoster is an acute disease the lesions of which are observed in the cutaneous distribution of one or more nerves. The eruption of herpes zoster is unilateral; the precise limitation of the eruption to one-half of the body is of great diagnostic significance (Musser). Pain is the most important subjective symptom. The pain is localized in the nerves in the distribution of which the eruption takes place. The pain may precede the eruption for several days, and persist long after the eruption subsides. The pain is severely neuralgic in character, and causes insomnia and depression.

Dr. Spiller, associate in the Neurological Department of University of Pennsylvania, considers it possible an herpetic type of poliomyelitis may be established, stating that much the same lesions are found in the intervertebral ganglia as in the spinal cord. (Spiller, "Diagnosis of Poliomyelitis," Pa. Med. Jour., Dec., 1911.)

Sixteen cases of herpes zoster occurred coincidently with the epidemic of poliomyelitis at Penryn, Cornwall, in the summer of 1911 (Gregor and Hopper). The majority of these 16 cases of herpes zoster occurred in elderly women. In many cases there was a severe pain and general malaise, and constipation was a marked feature. The eruption appeared on various parts of the body. Six had lesions on the neck, shoulder-blades, deltoid, and pectoral muscles; 4 over the intercostal muscles; 3 over the recti and buttocks; 3 on the lower limbs.

Chorea.—My attention was first drawn to the possibility of chorea being an acute infectious disease by Dr. M. C. Potter, of Rochester, who observed the onset of chorea in a previously healthy boy 10 years of age, while at an early summer camp. There was an acute febrile onset in this case. Dr. Potter further correlated the illness to the first exposure to the bites of mosquitoes during that season.

The most frequent manifestation of chorea is the involvement of a single neuron, usually one supplying a fiber of the facial nerve, with an involuntary twitching of the muscle motivated by that fiber. Such lawless and irregular twitching of the muscle may, however, involve an extremity; one side of, or the entire body. The disease shows a tendency to a spontaneous recovery, yet more often the patient is left with an ungovernable facial spasm for life.

Chorea is said to be transmitted (1) by imitation; (2) by heredity:—

1. The little girl who plays with a comrade who is a victim of this irritative lesion of some neuron or group of neurons, and who later develops a twitching of some muscle or group of muscles, is supposed to have developed the spasm voluntarily. The idea is preposterous. She is told to desist from the practice, and in a few weeks perhaps the spasm disappears. The mother considers that her admonition controlled the supposed mimicry. If the disease is not controlled, still the mother will state that it began from imitation of another case of chorea. It is stated in a medical textbook:—

"Thus one child sees another child with chorea, and, through imitation, *performs* the same irregular *involuntary* movement." (Herrick.)

2. Hereditary chorea is said to be a rare affection which the patient can usually trace back through several

generations. This is another instance, like tuberculosis, where it is difficult to displace an infection when it has once taken a firm grip on a family.

The etiology of chorea is as yet unknown, but the author considers it probable that this symptom is caused by the same organism that produces poliomyelitis. It may be that its mention here will induce observation as to its coincidental occurrence in epidemics of poliomyelitis, or even more directly in the families which have been attacked by frank cases of infantile paralysis.

VIII. RAPIDLY FATAL INSTITUTIONAL DISEASE.

For emphasis the eighth and last place (in this classification) is given to a lethal group, which may be hypothetical, but distinctly claims consideration.

Several authorities maintain that poliomyelitis is slightly or not at all contagious, stating that cases seldom develop in a hospital ward where others are present. While observing that such transmission is infrequent in occurrence, *the claim that it does not occur can no longer be made.*

Harbitz, of Norway, has published a report of the transmission of poliomyelitis to 2 women nurses in attendance in the poliomyelitis ward of the Christiana Hospital for Contagious Disease in August, 1911, both cases with a fatal termination. Other cases of transmission occurred in this hospital, and the facts are of so much importance, and have such a direct bearing on the subject of institutional poliomyelitis, that the report is here included:—

> As an argument against the contagiousness of poliomyelitis it has been advanced that nurses who take care of these patients escape the disease. This argument does not hold good; in a separate pavilion of the hospital for epidemic diseases at Christiana

were several poliomyelitic patients during July, August, and September, 1911. Two of their nurses, aged 28 and 29, became ill in the latter part of August and died with acute poliomyelitis. They had served in this hospital division for about one month. The virus must have been quite widely disseminated in this hospital, because in August there occurred in the diphtheria pavilion 4 cases of acute poliomyelitis, among children, of which 3 were in the same ward.

In the scarlet-fever pavilion 1 case of acute poliomyelitis occurred in a child who had previously been taken care of by one of the two nurses who died of poliomyelitis; and finally a nurse who served in one of the medical sections of the same hospital took the disease and died. In this last case it was impossible to trace the course of the infection. These cases, which occurred almost simultaneously, can be explained only by the existence of a direct infection and wide dissemination of the virus among adults and children. ("Poliomyelitis in Norway in 1911," Harbitz, Jour. Amer. Med. Assoc., Sept. 7, 1912.)

Here is a total of 8 persons, 3 nurses and 5 children, contracting epidemic poliomyelitis during a limited period of time while in residence in an institution; an institution, moreover, for the specific care of contagious diseases, where presumably all the modern technique of prophylaxis is known and rigidly enforced. Harbitz, in the same report, gives one more instance of the institutional occurrence of acute poliomyelitis, which he considers an instance of the latency of development of the virus:—

> A young woman who had been in solitary confinement for three months in the prison contracted a case of acute poliomyelitis; as hers was the only case inside the prison, it is reasonable to conclude that she came to the prison already infected. (*Ibid.*)

A not dissimilar case came to my attention in Wisconsin in 1909. A young man was committed to the county jail, which was also used as a lodging house for the floating tramp population run in for vagrancy. The second month of confinement the young man became acutely ill and de-

veloped a paralysis of the right arm. The young man's sweetheart informed me that there were vermin in his cell. *I am inclined to the opinion that each of these cases* resulted from transmission from cases of the arrested and unrecognized type, and may well have been occasioned by the agency of bedbugs. We do not yet know the means of transmission of this disease. If such transmission should prove to be by inoculation, we will say that, through the agency of cimex, in a well-ordered hospital it would never occur.

We are also ignorant of the degree of virulence which poliomyelitis may attain in the human host; in monkeys it has attained a mortality of 100 per cent. (Flexner, "Poliomyelitis," Jour. Amer. Med. Assoc., Sept. 24, 1910.)

Grant that a virus (whose usual strength shows a mortality rate of 15 per cent. and a disability rate of 60 per cent.) becomes suddenly and greatly enhanced in virulence *in vitro;* grant that the usual unknown inhibition of transmission of this virus is removed (or the agent of transmission is present), would we not look for a sudden acceleration of those destructive powers which have already earned from the public the name of the Children's Plague?

Such an acute epidemic asphyxia has been manifested in the past in England, with grave and alarming results which occasioned the publication of an official Blue Book. (R. Miller, "Acute Polioencephalitis," The Practitioner, London, April, 1910.) The cases tended to occur in small epidemics, in which after a few hours' illness death occurred suddenly with the signs of an acute asphyxia. The cases were supposed to be occasioned by a polioencephalomyelitis in which the vital centers in the medulla were the first to be attacked.

We still have in mind the sudden and unexplained deaths of 8 babies in an institution. A child with chicken-

pox was brought into this institution. The rash of poliomyelitis is frequently mistaken for that of measles and chicken-pox. The sister of this child and 7 other inmates suddenly died, with stiff neck, convulsions, and asphyxia. A young woman was indicted for their murder, but the charge was not sustained. Cervical rigidity, convulsions, and paralysis of respiration, as we have seen, are the cardinal symptoms of the acute fatal bulbar type of epidemic poliomyelitis. But the acute fatal bulbar type occurring here and there as isolated cases during an epidemic usually takes from three to seven days for its development.

It seems evident that for the kindling of this fire we must have not only the fuel and the match, but the fuel must have been rendered especially inflammable, to make ignition so nearly approach an explosion. To leave the figure of speech, children and adults in institutions live a highly artificial life; the child is susceptible to such crowding in inverse proportion to its age; the adult seeks the shelter of a lodging house with his resistance to disease impaired by undernourishment, exposure, and fatigue. It is under similar conditions that any transmissible disease may become suddenly malignant in its manifestations.

Poliomyelitis is proved to be a transmissible disease. It has been transmitted artificially from man to monkey through many series of such transmissions by inoculation. The virus of poliomyelitis, transmitted from man to the monkey made susceptible by a highly artificial mode of life, altered rapidly in potency, until the mortality rate in monkeys reached 100 per cent. (Flexner). The virus of poliomyelitis transmitted from man to man through a series of human hosts with lessened resistance and increased susceptibility, would tend to produce, under given circumstances, the form of poliomyelitis classified as type 8, the rapidly fatal institutional type under discussion.

The symptoms of the fulminating and rapidly fatal cases which occur in every epidemic of poliomyelitis may also be studied in relation to the foregoing: collapse; cold extremities; vomiting of partly digested blood; suffocation; cardiac paralysis.

TWO ATTACKS OF ACUTE POLIOMYELITIS IN SAME CASE.

Acute Anterior Poliomyelitis.—On a warm day in August, 1891, when 25 months old, after an indiscretion in diet, and without antecedent traumatism, Eshner's patient was seized with fever lasting three days, and associated with pain in the right leg and the back. There had been no vomiting and no diarrhea. On the fifth day the right lower extremity was found to be paralyzed, without apparent alteration in sensibility. The paralysis increased in severity for a week, and then it began gradually to diminish. The upper extremities, the left lower extremity, and the face were unaffected. The general nutrition was preserved, but the right lower extremity was moderately wasted. Intelligence was good and sensibility was unaffected. The gait was wobbling, the feet being held rather far apart in walking. There were no contractures and no deformity. The knee-jerk was normal on the left, enfeebled on the right. The circumference of the right leg was 7¼ inches, that of the left 7½ inches. The muscles of the right leg responded less well to faradic stimulation than did the muscles of the left leg, but there was no degenerative reaction. The patient occasionally had nocturnal enuresis, but there was no evidence of rachitis. Under treatment with massage and electricity for eight months practical recovery took place. Eleven years after this illness, in March, 1903, a day after a fall, resulting in injury to the left shoulder and the left elbow, the patient developed weakness in both hands, more marked in

the left. She had not been feeling well at this time and was "nervous," although she was attending school and had no fever or nausea or vomiting. While the symptoms manifested were those of acute anterior poliomyelitis, Eshner points out that someone might attribute them to peripheral-nerve injury in consequence of the fall. The development of the symptoms a day following and not immediately after the accident; the involvement of both hands primarily, even though in slight degree and but transitory in character, on the uninjured side; the absence of sensory alterations, certainly entitle the possibility of a spinal rather than a peripheral lesion—a poliomyelitis rather than neuritis—to serious consideration. (Jour. Amer. Med. Assoc., Oct. 8, 1910.)

RECRUDESCENCE OF POLIOMYELITIS.

Eckert, in the following quotation, gives a case of recrudescence:—

H. Sch. was taken sick September, 1903, with poliomyelitis, and was paralyzed in the left leg. Tenotomy was done and splints applied. At the beginning of April, 1909, occurred a second infection, and paralysis of the right leg.

"A positive case of this nature has come under my observation. In a family living in a typically rural part of Massachusetts sixteen years ago a girl, then 3 years of age, had an attack of poliomyelitis and recovered with a residual paralysis of the deltoid of the left arm. The family numbered, in 1910, 5 children—4 girls and 1 boy. In 1910 a girl 12 years of age became violently sick and died in a few days. The attack was thought to have been ptomaine poisoning, though characterized by many nervous symptoms. Early in August, 1911, the family went for a vacation to Maine. After three weeks' stay in Maine a 6-year-old girl was taken sick on August 24th and died

with respiratory paralysis in four days. No diagnosis was made by the attending physician.

"On August 28th, the boy, 3 years and 10 months old, was taken sick at Warren, Me., in a similar manner. There was, however, a slight paralysis of left face. The family then returned to Massachusetts, where the funeral of the girl was held.

"On August 31st, the oldest girl, 19 years old (who when she was 3 years of age had poliomyelitis), was taken sick and within a week developed a distinct Landry's paralysis, involving all four extremities, neck, back, abdomen, and respiration.

"On September 2d, a younger sister, aged 16 years and 9 months, was taken similarly sick. She developed rapidly the Landry type of poliomyelitis and died in a few days.

"This group of cases is doubly interesting from the fact that 4 cases occurred in the same family, and, further, that one of them, the 19-year-old girl, had had a previous attack of the same infection sixteen years before." (Sheppard, "Poliomyelitis in Springfield, Mass.," 1910.)

CHAPTER VII.

Diagnosis of Poliomyelitis in the Preparalytic Stage.

In any disease which is sufficiently serious to cause death or permanent disability within two to five days after its inception, it is apparent that treatment, to prevent such consequences, must be promptly instituted. Therefore in every disease of that character it is absolutely necessary that an early and correct diagnosis should be made. *Poliomyelitis is such a disease.* (McIntyre, Minnesota.)

Half a decade in America has added one to the brief list of diseases which the diagnostician must carry ever foremost in mind when called to attend a case of acute illness in child or adult. Poliomyelitis, because of its rapidly destructive or fatal lesions and its pandemic invasion of the country, supersedes typhoid, scarlet fever, diphtheria, or pneumonia, as well as all tortoise-footed diseases, in bedside cogitations. The period of time elapsing between the onset of the disease and the oncoming of paralysis is brief indeed. Deferring diagnosis until confirmation by oncoming paralysis confiscates the hours during which treatment modifies the course of the disease. And paralysis may become apparent only as a terminal asphyxia.

The symptoms and signs which warrant a diagnosis of poliomyelitis in the preparalytic stage are:—

I. Aura of onset.
II. Hyperesthesia a cardinal symptom.
III. Cervical tension a cardinal symptom.
IV. Hyperpyrexia, which declines by crisis.
V. Increased respiration rate with no lung findings.
VI. Pulse range 120 to 200.

VII. Basilar headache, considered intolerable.
VIII. Backache, considered intolerable.
IX. Knee-jerk exaggerated, diminished, lost, and unequal in 2 cases.
X. Vomiting.
XI. Constipation.
XII. Retention of urine.
XIII. Rash, measly or purpuric.
XIV. Epistaxis.
XV. Bulging fontanels.
XVI. Unbroken orientation and unimpaired judgment.

AURA OF ONSET.

The onset of the disease is the same in cases which later show cerebral or spinal localization, or pass into the arrested form of the disease; but such is the toxic affinity of the virus for the integers of the nervous system, that a neuropathic aura precedes the acute onset in a majority of cases. Dizziness, ataxia, and inco-ordination in the adult; tremor, quivering eyelids, stumbling, and unaccustomed falls in the child are the frequently observed warnings of the oncoming of the onset of this disease.

HYPERESTHESIA.

Hyperesthesia is the characteristic diagnostic symptom of onset of poliomyelitis. This hyperesthesia is always present at some level of the spine; usually cervical, it may extend down to the sacrum; it is also found present in that area of the torso or extremities which will later be paralyzed. This hyperesthesia is almost intolerable; the physician will be amazed at the agonized resistance to handling of some child who has hitherto regarded him as a friend; the pain is so exquisite as to wring from a grown man the wish that his arm might be cut off; the hyperesthesia of

the muscles of the shoulder-girdle has prompted a diagnosis of dislocation of the shoulder; a child will scream with the pain in the preparalytic leg when placed on the bedpan; Müller reports a case in which a diagnosis of appendicitis was occasioned by the hyperesthesia of the right abdominal parietes subsequently paretic, and considers that this symptom alone is sufficient for diagnosis in the illness of a hitherto healthy child. This hyperesthesia appears to be a severe myalgia, and is also frequently mistaken

Fig. 60.—Profound stupor, paralysis of left arm, retraction of neck. (After *Peabody, Draper,* and *Dochez.*)

for rheumatism. The cause of the hyperesthesia is the involvement of both anterior and posterior spinal ganglia, where fibers of pain, temperature, and tactile sense affiliate with fibers of the sympathetic, and then fuse with motor conduction fibers.

CERVICAL TENSION.

A stiff neck is the constant accompaniment of onset; it is usually associated with basilar headache and backache; the extensor cervical muscles are spastic and the rigidity is demonstrated by flexing the head on the chest; in one of Shidler's cases, a 10-year-old girl, the initial

symptom was an inability to see down a posthole when she stood close to it because of the stiffness of the neck muscles. If the child is told to place the chin on his chest he will do so by dropping the jaw. The rigidity is anteroposterior; lateral motion of the head is possible in those cases in

Fig. 61.

Fig. 62.

Intercostal paralysis, protrusion of abdomen, and retraction of thorax. (After *Peabody, Draper,* and *Dochez.*)

which there is no involvement of the sternomastoid. If there is a considerable spasticity the head will be retracted, the chin well up if the child is going about, or noticeably extended on the pillow; stooping forward is so painful the child may refuse to drink, according to Anderson, or the

nursling to nurse. Armstrong reports a case of a 5-year-old boy who for one week laid on his face "because it hurt his neck to lie on his back." The entire spine may be spastic, with the torso rigid as a board. Opisthotonos is not uncommon. Convulsions are fairly frequent in young children, and in at least 1 case, a young male of 20 years, convulsions were severe and continued for twenty hours, resulting in death.

HYPERPYREXIA.

A high fever, which may be preceded by a chill and accompanied by severe sweating, is characteristic of the onset; Draper and Peabody consider it the most characteristic of all symptoms. The rise in temperature is invariable, but frequently overlooked. The fever declines by crisis, with the amelioration of other acute symptoms preceding the onset of the paresis or paralysis. With involvement of the heat centers in the ascending type the fever may rise to 107-110° F., rectal record; these cases suggest heat stroke and have been mistaken for heat prostration.

INCREASED RESPIRATION RATE.

The respiration rate is about doubled and may be trebled at onset; there are no lung findings, and the tremendous increase in the number of inspirations is very puzzling until it is recalled that the centers for respiration and the phrenic nerve are comprehended in the cervical plexus; the constant cervical tension of onset would indicate that the cervical spinal nerves are first involved in all cases. This hurried breathing is early; later, with the onset of paralysis in thoracic and diaphragmatic fibers, the breathing becomes shallow and of the reversed abdominal type, in which the upper abdomen is sucked in during inspiration.

INCREASED PULSE RATE.

There is a marked increase in the pulse during the early irritative stage. In children the pulse may range from 120 to 200; in adults, to 120 or higher. This fact is often not mentioned by other authorities, and many of them omit to mention cardiac action altogether; it may be that this is due to the frequent omission by the examiner, owing to some difficulty in obtaining a child's pulse rate, of a pulse record in these cases. In Dakin's careful record of the Mason City, Iowa, cases of poliomyelitis the pulse of onset is given in 31 of the 36 cases seen:—

Age 1 year.	1 case.	Pulse 180.
Age 2 years.	2 cases.	Pulse 130-150.
Age 3 years.	8 cases.	Pulse 112-120-120-120-130-130-130-140.
Age 4 years.	2 cases.	Pulse 175-180.
Age 5 years.	2 cases.	Pulse 120-150.
Age 6 years.	3 cases.	Pulse 140-150-190.
Age 7 years.	3 cases.	Pulse 120-130-170.
Age 8 years.	2 cases.	Pulse 120-150.
Age 12 years.	1 case.	Pulse 120.
Age 16 years.	1 case.	Pulse 120.
Age 26 years.	1 case.	Pulse 100.
Age 30 years.	2 cases.	Pulse 80-100.
Age 42 years.	1 case.	Pulse 100.
Age 44 years.	1 case.	Pulse 100.
Age 48 years.	1 case.	Pulse 90.

(Rearranged from Dakin, "Poliomyelitis," Iowa Med. Jour., Nov., 1910.)

From series of Dr. E. S. Hayes, Eau Claire, Wis.:—

Sept. 12, 1908. Mary M., aged 4. Temp., 102° F.; pulse, 140; paralysis. July 23, 1908. Baby W., aged 2. Temp., 103° F.; pulse, 130; paralysis. Sept. 19, 1908. Emil K., aged 8. Temp., 101° F.; pulse, 130; paresis. (Personal communication to Dr. Manning.)

REFLEXES DURING ACUTE STAGE.

The first effect of the irritative stage is to augment the knee-reflex; it may be exaggerated in both knees; usually an exaggerated reflex is obtained in the more hyperesthetic leg, with no change in the reflex of its fellow; later there will be a diminution of the reflex or it may be lost in the leg first affected, while at the same time the reflex of the second leg will now show augmentation. Brudzinski has demonstrated two valuable reflexes which are found in children with acute disease of cord and brain: (1) The contralateral reflex of the legs consists in a concomitant reflex of the leg on one side when passive flexion of the leg on the other side is made. This sign has been found useful in the diagnosis of meningitis. (2) The Brudzinski neck sign is obtained when passive flexion of the neck forward produces flexion of the legs at the hips and knees, and also flexion of legs on pelvis. This reflex, which indicates the presence of meningitis, would need to be attempted with much gentleness in poliomyelitis or otherwise would occasion much suffering.

VOMITING.

Vomiting is a constant accompaniment of onset; it is a part of the paresis of digestion responsible for the accompanying condition of constipation. The case may vomit once, or repeatedly. When projectile vomiting is a feature of onset the case is usually of the cerebral type.

CONSTIPATION.

Constipation is a feature of onset of poliomyelitis in this country; in Germany an onset with diarrhea has been noted so frequently as to be recognized as characteristic. The constipation met with here is really an obstipation, and

is usually associated with difficulty in urination or retention of the urine, an excellent indication that both conditions are due to partial paresis.

RETENTION OF URINE.

Delayed urination is the rule and retention of urine frequent in the initial stage. Prompt efforts to secure elimination may allay this condition, but dysuria, retention with overflow, involuntary urination and anuria have all been observed to occur. A distended bladder was diagnosed as a case of intussusception and operated, a subsequent paralysis clearing diagnosis.

SWEATING, EPISTAXIS, AND CUTANEOUS RASH, OR PURPURA.

Sweating of a severe character has been reported as characteristic by some observers and ignored by others. It is possible that sweating is a symptom which, like the marked increase in the pulse rate, occurs so early that it is missed by the attendant. Müller considers the profuse sweating may be due to the involvement of spinal sweat centers. I am now of the opinion that the profuse sweating, as well as the occasional epistaxis, and the more than occasional rash, are varying manifestations of the pathogenic permeability of the blood-vessel walls; these systemic manifestations of mechanical permeability, varying in degree from leakage to hemorrhage, correspond to the process which is known to be taking place in the walls of the spinal blood-vessels.

EXAMINATION OF SPINAL FLUID.

A quantitative increase in the spinal fluid is shown by symptoms of pressure, bulging of the fontanels, Macewen's tympanitic note, and accelerated flow on lumbar puncture.

The spinal fluid is clear, becoming slightly opalescent at the paralytic stage, is sterile, and is increased in quantity and globulin content.

CEREBROSPINAL FLUID. SOPHIAN'S TABLE.

	Normal	Meningism	Poliomyelitis Polioencephalitis	Cerebrospinal Meningitis	Streptococcus Pneumococcus Influenza, etc. Meningitis	Tuberculous Meningitis
Color	Clear	Clear	Clear, occasionally bloody, from fresh or old hemorrhage	Cloudy, pus sediment	Cloudy, pus sediment	Clear, white flakes, fibrin-network
Pressure	Low, escapes slowly drop by drop	+	+ to + + to + + +	+ + to + + +	+ + to + + +	+
Quantity	Little, few c.c.	(Up to 50 c.c. or more)	(Up to 50 c.c. or more)	(Up to 100 c.c. or more)	(Up to 100 c.c. or more)	(Up to 100 c.c. or more)
Cytology	Few cells, leucocytes and endothelial	Few cellular elements	Cells increased in number Lymphocytes 90% or more	Cells numerous (polynuclear up to 100%)	Cells numerous (polynuclear up to 100%)	Cells numerous (lymphocytes up to 90%)
Bacteriology	Sterile	Sterile	Sterile	Meningococcus	Infecting organism	Tubercle bacillus
Albumin (nitric acid test)	Faint trace	Trace	Trace to +	+ + +	+ +	+
Globulin test	Negative	Negative	Positive in early stages	+ + +	+ + +	-

OUTLINE FOR SERUM EXAMINATION. (DU BOIS.)

Number of cubic centimeters of spinal fluid.
Clear. Cloudy. Fibrin.
Cytology.
Bacteriology. Spread. Culture
Globulin. (Butyric acid test.)
Albumin. (Nitric acid test.)
Clinical diagnosis.
Animal inoculation.

HISTORY OF EXPOSURE.

The prevalence of the disease in the community should always be considered; a history of exposure is strong presumptive evidence of the presence of the disease; no polio-

myelitis in community does not negative the disease; old cases in the house are to be considered in the light of possible carriers.

CEREBRAL TYPE.

Many unrecognized cases of the cerebral type occur, especially among adult males. In a group of 20 cases seen by Sophian in three weeks' time during the fall of 1911, there were 12 cases of the cerebral type, an astonishing percentage, which illustrates anew the fatuity of considering the spinal myelitic type to be typical of this disease.

The usual picture of the cerebral case is a spastic hemiplegia with oculomotor or facial involvement. Facial paralysis or an internal strabismus may be the only manifestation.

The spastic hemiplegic form in the adult is clearly differentiated from cerebral hemorrhage by the aura of onset, chill, high fever, hyperesthesia, and, most important of all, the unbroken orientation for time and place, with no loss of consciousness.

CHAPTER VIII.

Differential Diagnosis.

If we think of the disease as a poliomyeloencephalitis with meningeal complications and appreciate the fact that the disease may affect any or all parts of the gray matter of the central nervous system, and in all possible combinations, we shall understand better the innumerable manifestations of the disease and be less likely to overlook it or mistake it for some other condition. We must never forget how variable the symptoms may be. If we are awake to all the possibilities, we are not likely to overlook it. (Morse.)

Meningitis.—Epidemic cerebrospinal, tuberculous, suppurative. Meningism is one of the diagnostic signs of the onset of infantile paralysis. It may vary in degree from a slight nuchal tenderness and rigidity to the most extreme type of meningeal irritation, the meningeal form of the disease. The diagnosis is usually made clear by the appearance of paralysis. Cases appear coincidently with the epidemic, with meningitic symptoms and without paralysis. Wickmann found, clinically and by autopsy, that such cases ran the "whole course as a meningitis serosa."

It is of great importance to make an early diagnosis. When the case simulates an acute meningitis, or a tuberculous meningitis, lumbar puncture, with an examination of the spinal fluid, is the ready means of positive diagnosis.

In a case of infantile paralysis puncture will be followed by a rapid drop flow, or even spurting of the spinal fluid. Ten c.c. is sufficient for purposes of examination. The fluid will be found clear, or exhibiting a slight shimmering or opalescence in the preparalytic stage. On examination it will be found to be sterile. Lymphocytes, small and large, make up 90 per cent. of the white cells, with a very few

polynuclear leucocytes. The supernatant fluid, after centrifuging, will give a marked protein reaction with the Noguchi butyric acid test.

Fig. 63.—Familial type. Patient one of three brothers equally afflicted. This case steadily improving under muscle re-education. (N. Y. Hospital for Deformities and Joint Diseases.)

Tuberculous meningitis will be indicated by a flocculent fluid. If fluid is allowed to stand twenty-four hours, a fibrin network will form and be plainly demonstrable.

With Heller's test for albumin, in tuberculous meningitis, the percentage of albumin will be high and a thick cloud will form. In the spinal fluid of poliomyelitis there will be only a faint reaction to this test.

In tuberculous meningitis the bacillus may be demonstrable, but this is so uncertain that its absence cannot be considered a diagnostic point.

In epidemic cerebrospinal meningitis the meningococcus will be found in the centrifuged sediment of the spinal fluid.

In suppurative meningitis pus will be present in the fluid.

In streptococcus meningeal infection following middle-ear disease there will be a clear history of the exciting cause.

"Meningism may complicate any acute contagion, especially bronchopneumonia. Here the differential diagnosis is dependent on the previous history of the case." (Sophian.)

Epidemic Cerebrospinal Meningitis.—Early in an epidemic of poliomyelitis, the cases of an encephalic type are mistaken for cases of epidemic cerebrospinal meningitis; and this is not surprising, for the symptoms of the vicious onset are the same. Koplik suggests as a point of distinction that in polioencephalitis there is a short preliminary period in which the patient, having had a high fever, continues to be about. Vomiting, basilar headache, cervical rigidity, high temperature, and delirium, with or without convulsions, usher in the attack in both instances. Isolation and quarantine are recommended while making a study of the case. Without examination of the spinal fluid, it may be impossible to form a diagnosis during the first forty-eight or seventy-two hours. The second or third day, the characteristic paralysis is apt to appear in cases

of poliomyelitis. Local palsies are rare in epidemic cerebrospinal meningitis, and transitory and late with high temperature.

The temperature in epidemic meningitis is high at onset and remains high with an intermitting curve. The temperature of poliomyelitis is high at onset and declines by crisis before onset of paralysis.

The pulse of poliomyelitis is extremely rapid in the preparalytic stage, ranging from 110 to 200. The pulse of epidemic meningitis has more of the characteristics of the compression pulse.

Delirium may be marked, especially in sleep, but will lessen with the improvement of other symptoms in the one disease; in the other the delirium, though broken by lucid intervals, may progress to a stuporous condition, and coma.

The spastic spine in cerebrospinal meningitis is pathognomonic. The head is fixed and immovable. In poliomyelitis, while the rigidity is marked, yet the patient himself can turn his head from side to side.

Kernig's sign will give a similar reaction in each leg in cases of cerebrospinal disease, while in infantile paralysis there will be elicited a much more spastic condition in one leg than in the other.

Suppurative Meningitis.—In meningitis secondary to pneumonia, bronchopneumonia, middle-ear disease, or the meningism which may complicate any acute contagion, there will be a history of the preceding trouble.

The common complication of middle-ear trouble following influenza or grippe should be kept in mind, for it may precede a meningitis.

Tuberculous Meningitis.—In tuberculous meningitis the condition is secondary to a previous infection, and the history of individual and family is to be considered. It occurs alike in infants, adolescents, and young adults. In

poliomyelitis a sudden acute onset will ensue after a previous state of perfect health. In tuberculous meningitis there is a gradual onset, with a history of some days or weeks of malaise and temperature. In tuberculous meningitis there may be delirium, convulsions, followed by coma; this also may be seen in poliomyelitis, but in the former case apathy will be marked, while in infantile paralysis there is an alert, strained look, and if the patient falls into a semistupor he will rouse at once to answer a question; will, in fact, react to his surroundings and complain of many disturbing factors, such as a ray of sunlight, a noise on the street, the jogging of his bed, to which the sufferer from tuberculous meningitis is quite indifferent.

In tuberculous meningitis the spinal rigidity is not extreme, it may be elicited on examination. In poliomyelitis, the child may refuse to drink, from the pain engendered. A little girl couldn't "manage" a lemonade straw, and it was only after considerable effort and the bending of a glass tube that she could be induced to take the iced drinks she was craving. The nursling will not always nurse for the same reason; the bending of neck or any movement of head is too painful.

In both diseases hydrocephalus may develop, with bulging of the fontanels. Cerebral palsies are also seen. Local palsies in tuberculous meningitis are usually transitory and shifting. They are said to be due to the shifting of the fluid in the ventricles, and may occur in face, arms or legs. The paralyses of poliomyelitis are not shifting or transitory; some mild cases develop only a paresis, which rapidly clears, but these cases are of so mild a character that there is no danger of confusing them with tuberculous meningitis.

The temperature in tuberculous meningitis is characteristic; afternoon rise to 103° F., and in the late stages a high, intermitting range. Poliomyelitis of the cerebral

type begins with a high temperature, which rapidly subsides, and remains within one degree of the normal throughout the illness.

The mental state of the patient in the polioencephalic form of poliomyelitis is perhaps the most decided diagnostic point. The delirium or stupor, if present, lightens, and the patient proceeds to recover. They do not seem to be suffering from brain disease. A case in the Connecticut epidemic, with a complete hemiplegia, developed an aphasia on the fifth day, but "looked bright." (Sophian.)

Meningism in Infectious Diseases.—Meningeal phenomena may supervene as the sequence of toxic influences on the central nervous system, with no change in the meninges. Typhoid fever, pneumonia, and other acute infectious diseases may produce this intoxication, which is manifested as a meningism. There is no change in the spinal fluid, nor demonstrable alteration in the meninges.

Suppurative Meningitis.—In meningitis secondary to pneumonia, bronchopneumonia, middle-ear disease, or the meningism which may complicate any acute contagion, there will be a history of the preceding trouble. The common complication of otitis media following influenza or grippe should be kept in mind, for it may precede a meningitis.

DIFFERENTIAL DIAGNOSIS.

	Poliomyelitis, cerebral type, with or without meningitis.	Cerebrospinal meningitis.	Tuberculous meningitis.
Season	Maximum, late summer.	Winter and spring.	
History[1]	Presence of epidemic in community. Hist. of exposure.	Presence of epidemic.	Tub. infection of patient or family.

[1] The history of suppurative meningitis is: Otitis media; mastoiditis; sinus infection.

DIFFERENTIAL DIAGNOSIS (*Concluded*).

	Poliomyelitis, cerebral type, with or without meningitis.	Cerebrospinal meningitis.	Tuberculous meningitis.
Nutrition	Healthy, robust and active.	Impaired.	Impoverished.
Onset	Acute.	Acute.	Gradual (early stage slow).
Pulse	Rapid, 120-200.	Slow.	Moderately rapid.
Temperature	High, 1 to 4 days, declining by crisis as paralysis appears.	High from onset; intermitting curve.	Characteristic P.M. rise; high, intermitting range in late stages.
Mental state	Excitable, staring.	Stuperose.	Apathy marked.
Bowels	Paresis and obstipation.		
Skin	Multiform rash.	Rash. Herpes.	No rash. No herpes.
Cervical rigidity.	Present, but can move head from side to side.	Head fixed and immovable.	Not marked.
Kernig's sign	One leg much more spastic than other.	Equal in the two legs.	
Local palsies	Characteristic; regressive but not transitory. Head, trunk, or extremities.	Rare. Face or eyes only.	Transitory of face, arms, legs.
Delirium	During sleep and ceases with oncoming of paralysis.	With lucid intervals.	Delirium, followed by convulsions and coma.
Tremor	Marked.		
Spinal fluid	Increased pressure. No organisms. Clear or a slight shimmering. No fibrin clot. Hypoleucocytosis. (Lymphocytes 90%.) Albumin, a trace, and protein.	Meningococcus. Turbid. Albumin.	Koch's bacillus. Turbid, flocculent. Albumin % high. Fibrin % high.

Other Diseases of the Cerebrospinal Axis, With or Without Paralysis.—The confusing nomenclature of acute nervous diseases varies widely in the accepted textbooks. It is evident to the mere student pathologist that indicative symptoms have been classified and described as separate diseases. The cerebrospinal axis is the most highly developed and specialized organ known to man. A large

Fig. 64.—Facial paralysis in 23 months' infant referred as a case of infantile paralysis. Father died at 26 years. Infant developed typical syphilitic eruption on legs. (N. Y. Hospital for Deformities and Joint Diseases.)

Fig. 65.—Acute bulbar type with paralysis of facial nerve. (N. Y. Hospital for Deformities and Joint Diseases.)

class of the manifold lesions, ranging from a mild pressure edema to necrosis, which may occur in any one of its intricate parts, disturbing one, or any possible combination, of its functions (which govern and induce every voluntary and involuntary act relating to life), may have been incited by one unknown destructive infection.

Friedreichs's Ataxia.—"Often attacking several children in the same family." May prove to be hereditary only in the sense with which today we explain how pulmonary tuberculosis appears and reappears in a family, from successive series of reinoculations. This "hereditary disease" develops most frequently in childhood and at the age of puberty, or during the susceptible period for contracting an infection.

Paralysis Agitans.—A tremor of the extremities in the aged following "the exhaustion of an acute disease," and succeeded by rigidity, contractures, and atrophy. What would result from the shock of an infection of the cerebro-spinal axis not sufficiently grave to paralyze or kill? Would it not have a tendency to precipitate and exaggerate those symptoms of senility, tremor, ataxia, and inco-ordination in men or women well past life's meridian? And does it not leave ample proof of its antecedent cause in the muscular weakness, rigidity, contractures, and atrophy?

Acute Transverse Myelitis.—Ushered in by fever, headache, delirium,—abruptly with a convulsion,—by rheumatoid pains,—retention of urine (a very important and early symptom),—and a possible spastic paraplegia, form so clear a group picture of the onset and progress of a case of poliomyelitis with lumbar involvement only, that a differential diagnosis is hardly in order. Landry's, or the acute ascending paralysis of the textbooks, is now known to be a mere variation in the mode of attack of poliomyelitis epidemica acuta.

It is questionable whether myelitis is essentially different in etiology from poliomyelitis. The symptoms differ merely because in the former more of the transverse area of the cord is involved. I have observed myelitis in an adult which developed last year during the epidemic of poliomyelitis, and it has occurred to me that possibly this case was a manifestation of poliomyelitis. (Spiller.)

Diseases Presenting Paralytic Conditions:—

Diphtheritic paralysis—diphtheria.
Syphilitic pseudoparalysis.
Tuberculous spondylitis with paralysis.
Obstetrical paralysis.
Hysterical paralysis.
Pseudoparalysis of scurvy.
Occupation neurosis.
Progressive muscular atrophy.

Diphtheria and Diphtheritic Paralysis.—The onset of acute epidemic paralysis is sometimes announced by a pharyngeal angina of so severe a degree that a diagnosis of diphtheria has been made.

Lillian B., Eau Claire, Wis., 10 years old, September, 1908 (during epidemic); membranous sore throat. Antitoxin given. At the end of one week of fever, delirium, prostration, right arm became paralyzed, then left; paralysis gradually extending to muscles of respiration, patient died on the twelfth day of illness. Diagnosis changed to infantile paralysis on appearance of characteristic paralysis. (Ashum.)

Diphtheria.—Enlarged cervical glands, absence of knee-jerk, slower pulse at onset (100 to 120), membrane attached, of appreciable thickness, Klebs-Löffler bacillus. Paralysis postdiphtheritic, manifested by nasal intonation, regurgitation of liquids through nose, and inco-ordinate movements, which appear late.

Epidemic Paralysis.—No enlargement of cervical glands; exaggerated knee-jerk; rapid pulse at onset (120 to 200), patches on throat isolated and superficial; paralysis usually of extremities first.

In true multiple neuritis the course of the disease is slower, the early febrile stage is absent, the palsy is usually symmetric and greater in peripheral parts. Poliomyelitis may begin with angina and thus suggest diphtheritic neuritis, but the paralysis follows the angina much later in diphtheritic multiple neuritis. There is not likely to be paralysis of the soft palate and of accommodation, or cardiac symptoms in poliomyelitis, and these are common after diphtheria. Multiple neuritis is believed by some to be more common in children than has been taught. (Spiller.)

Syphilitic Pseudoparalysis.—Absence of knee-jerk is characteristic of locomotor ataxia, and appears before the

inco-ordination of gait; there is also a preceding history of venereal infection, and gradual onset.

Epidemic Paralysis.—The knee-jerk is exaggerated during onset of disease. Confusion may arise, when an adult male with an admitted venereal infection, dates a slowly progressive ataxia, inco-ordination, and mental bewilderment from a certain "spell" of two days' illness occurring when acute paralysis was epidemic in community.

Luetic pseudoparalysis of infancy is due to an acute epiphysitis, and will be associated with characteristic skin lesions, etc., which will yield to mercurial treatment, and confirm diagnosis.

The general consensus of opinion is that syphilis is not a general factor in the upper neuron paralysis of childhood, and this opinion appears to be based on very good grounds. Nevertheless, a reconsideration of the part syphilis plays may be instructive and appears to be desirable for two reasons: First, because, in some cases at least, a permanent and crippling disability of both body and mind may be prevented by an intelligent anticipation of events; and, secondly, because, thanks to the methods of diagnosis perfected of late years, it may yet turn out that syphilis is a more important factor in the production of these paralyses than many have suspected.

Tuberculous Spondylitis With Paralysis.—The gradual onset of the condition, and the vertebral knuckle are characteristics of tuberculous disease of the spine which has advanced to the stage of pressure paralysis. Paralysis may be transitory when collateral pressure edema is relieved.

Obstetrical Paralysis. — "Obstetrical paralyses are limited to traumatized nerve-trunks, and are found usually in the parts likely to be handled with violence by the obstetrician—namely, the shoulders and arms." (Hummel.)

These cases when brought to the specialist usually give a clear history of birth palsy following difficult labor. If the labor was not protracted nor otherwise difficult, it would be well to keep in mind the possibility of an intra-uterine poliomyelitis or encephalitis.

Hysterical Paralysis.—Poliomyelitis in its chronic form may be simulated by an hysteria, "but the symptoms in purely functional cases do not group themselves usually in a manner typical of infantile paralysis." Hysterical deformity or contracture can be temporarily overcome by anesthetizing the case.

Pseudoparalysis of Scurvy.—This condition is, happily, extremely rare in the Northern States and usually institutional. According to Fenner, of New Orleans, this condition is not a rare disease among artificially fed children of the well to do in the South, and "paralysis is another common mistake in diagnosis." The differential diagnosis would relate to the hemorrhagic and spongy gums, hemorrhages into the orbit, hematemesis, bloody urine, and the immediate response to antiscorbutic diet: fruit juices, oranges, lemon, pineapple and fresh food.

Muscular dystrophy could be considered only in the late stages of poliomyelitis, and at this period confusion is possible. I have seen cases in which the differential diagnosis was difficult. (Spiller.)

Acute Infectious Diseases Without Paralysis:—

Enteritis.	Measles.
Typhoid.	German measles.
Rheumatism.	Scarlet fever.
Tonsillitis.	Chicken-pox.
Influenza—summer grippe.	Tetanus.
Pneumonia.	Rabies.
Pleurisy.	

Summer Diarrhea—Enteritis, Cholera Morbus.—The following table, compiled by Dr. H. W. Hill, epidemiologist, Minnesota State Board of Health, which he denominates an "interesting analogy by contrast," is given in the belief that nothing could more clearly demonstrate the lack of relationship between infantile enteritis and epidemic paralysis:—

	Summer diarrhea.	Anterior poliomyelitis.
Weather	Hot, damp.	Hot, dry.
Onset	Slow.	Abrupt.
Age	Under 2 years.	Over 2 years.
Feeding	Overfeeding.	Underfeeding.
Bowels	Diarrhea.	Constipation.
Previous health	Poor.	Good.
Incidence	Slum dwellers.	Rural dwellers.
Elimination	Marked.	Retention.
Dietetic errors	Continuous.	Accidental or absent (except deficiency).
Intestinal incidence	Marked.	Slight.
Nervous incidence	Slight.	Marked.
Relation to milk	Obvious.	None.

The following death certificates are taken from the records of Eau Claire, Wis., during the period poliomyelitis was epidemic there, July, August, and September, 1908. In each case there was one or more cases of clearly marked paralysis in house or immediate neighborhood:—

	Cause of death.
July 7th, Male, 1 year 4 mos.,	"Convulsions due to enteritis."
July 24th, Female, 7 years,	"Congestion of brain."
Aug. 16th, Male, 9 years,	"Paralysis of heart."
Aug. 28th, Male, 18 mos.,	"Acute indigestion."
Sept. 5th, Male, 4 years,	"Enterocolitis, congestion of brain."

It seems probable that in other communities a certain percentage of deaths due to poliomyelitis are thus credited. Vomiting and prostration are the only symptoms common to both. The seasonal occurrence is identical, but this probably indicates only that the transmission of the two diseases is dependent on insect carriers, whose numbers increase largely in summer.

	Typhoid fever.	Poliomyelitis.
Onset	Insidious.	Acute.
Season	Fall.	Summer.
Fever	Continuous.	Early fall by crisis.
Rash	Rose spots on abdomen.	Scarlatiniform, blotchy, or petechial and pustular over trunk and extremities.
Bowels	Diarrhea, pea-soup stool.	Constipation.
Appetite	Anorexia.	Hungry when temperature lowers, 3-4 day.
Pulse	Slow pulse characteristic.	Much augmented, 100 to 200.
Blood	Widal reaction.	Widal negative.
Paralysis	None.	Characteristic.
Spasticity	Not present.	Cervical or entire spine.

The early symptoms which might lead to a tentative diagnosis of typhoid fever in a case of poliomyelitis are the epistaxis, the extreme prostration, temperature, delirium, tremor, twitching and severe continued headache, and meteorism.

A rapid pulse, nuchal rigidity, and constipation would speak almost certainly for poliomyelitis. Season and presence of epidemic in community should be considered.

Paratyphoid.—The mode of onset alone would indicate diagnosis.

Rheumatism, Acute, Articular, and Muscular.—In the carefully investigated symptomatology of 19 cases of poliomyelitis, Dr. Shidler reports (Nebraska State Medical Association) that all but 1 case suffered from general tenderness. This agonizing tenderness, taken with the fever and prostration, probably accounts for the mistaken diagnoses of rheumatism. In rheumatism there is often a history of previous attacks which salicylates have re-

lieved; there is swelling and redness of the joints, and the pain in the joints is severe in the early stage. In poliomyelitis the pain corresponds more nearly to the distribution of great nerve-trunks, and increases after the fever begins to decline. There is not usually any swelling of the joints in poliomyelitis, yet this has been noted by a few observers.

The affected limb in poliomyelitis is cold to the touch, and distinctly cyanosed, assuming a dull-reddish, purple hue.

Muscular Rheumatism.—A localized myalgia, such as torticollis, pleurodynia or lumbago, usually appears as a spontaneous symptom or condition unrelated to an attendant or subsequent train of illness. In torticollis the head will be inclined toward the affected sternocleidomastoid muscle, and the spasm will disappear with catharsis and the application of heat.

Tonsillitis.—Cases have been diagnosed as tonsillitis due to swelling and inflammation of the tonsils, which, in an exceptional case, was reported extreme. If the mucous membrane of nose and throat forms the point of attack for the virus, it is not strange that the tonsils sometimes react markedly; this is the exception and not the rule.

A true tonsillitis will usually give a history of susceptibility and repeated attacks, with allied rheumatic conditions. The meningism that accompanies a severe attack of tonsillitis will be confusing. This meningism usually clears promptly on the exhibition of calomel.

	Tonsillitis.	Poliomyelitis.
History	Repeated attacks.	None.
	Rheumatic attacks.	None.
Onset	Chill.	Rare.
Tremor	None.	Usual.
Rash	None.	Usual.

Influenza—Summer Grippe.—Influenza, grippe, and the balance of the respiratory diseases reach their period of maximum incidence in February and March. When they appear during the summer they usually follow a period of damp and cold weather and are more apt to affect adults. The coryzal onset of influenza is rarely seen in poliomyelitis. Dr. C. A. Anderson, of Stromsberg, Nebraska, reports "complete absence of acute catarrhal trouble in the respiratory tract and eyes," in an analysis of 279 cases in the Polk County epidemic.

Pneumonia, Bronchopneumonia.—Rapid and shallow respirations, together with the abrupt onset, high temperature and racing pulse, suggest an impending bronchopneumonia when the child is first seen. There is sometimes an accompanying bronchitis, but this is rare. Physical examination will show the lungs are clear.

The respirations may reach or exceed 60 per minute for the first day of onset, with no apparent cause, and then slow down to normal; this may occur in the ordinary lower-segment type paralysis with no extension, or the mild cases with paresis only. This acceleration may, however, announce the onset of a case of the ascending type of paralysis with beginning involvement of the muscles of respiration.

Measles, German Measles, Scarlet Fever, Chicken-pox.—Skin rashes which follow the fastigium of poliomyelitis may simulate the eruption of any one of the acute exanthemata. A scarlatinal blush, a fine petechial rash, measly blotches, and a papular and vesicular eruption have all been observed. The measly rash with blotches somewhat smaller than in measles is most common, and has doubtless been efficient in scattering the infection of poliomyelitis far and wide. Many mothers are convinced that it is better for children to "take" measles when they are young, and are not averse to allowing them to play with supposed cases of

measles. In German communities there are always gute Hausfrauen who are considered sufficiently accurate diagnosticians for such trifling ailments.

Measles will have a history of an acute coryza with watering eyes, cough, diarrhea (usually),—in fact, an acute catarrhal inflammation of the mucous membranes of digestive and respiratory tracts; while in poliomyelitis in an early stage the eliminations are *nil*. In severe cases of measles, especially in adults, there is a pronounced meningism at onset, followed by an apathetic typhoid condition. The meningitis of poliomyelitis is of a much more pronounced type; the spastic spine, augmented reflexes, basilar headache, will serve to differentiate the two diseases; a history of the epidemic appearance of one or both in the community must be considered.

The rash of measles usually appears on the face, at the edge of the hair and back of the ears; the rash of poliomyelitis, on trunk and extremities.

German Measles.—The eruption of German measles also appears usually on the face and below hairline. There is an accompanying coryza, and enlargement of cervical and occipital glands, which will serve to differentiate it from poliomyelitis.

Scarlet Fever.—There is little danger of confusing a typical case of scarlet fever, for the brilliant scarlet blush appears early, usually at the lapse of twelve hours. The rash of poliomyelitis, which may assume the scarlatinal type, appears late, as the fever is declining.

Chicken-pox.—The diagnosis in the following cases was carefully reconsidered on the appearance of a pustular rash. The cases occurred during the apogee of the epidemic of 1908 in Eau Claire, Wis., and were seen in consultation:—

B. and W., brothers, aged 2 and 7 years, taken sick forty-eight hours apart; marked prostration, twitching, abolished reflexes, inability to raise head, moderate fever (102°); rapid, running, feeble pulse; entirely conscious. Paralysis of respiration terminated both cases the succeeding Sunday afternoon, the third and fifth day after attack, the baby dying at 3 P.M. and the older boy at 7 P.M. The pustular rash appeared all over torso and extremities of babe. The environment was most unsanitary.

Tetanus With Convulsions.—A poliomyelitis of Landry's descending type might closely simulate an attack of lockjaw, and the history of a recent slight trauma would tend to confuse the diagnosis.

S. M., of Wisconsin, 14 years of age, was present in a group of other boys, on the morning of the 4th of July, when a toy cannon exploded. He was struck by one of the flying particles and sustained a slight laceration. Late in the month he became ill with convulsions, and a diagnosis of tetanus was made by the attending physician. Antitetanic serum was telegraphed for, but paralysis of all extremities followed before it could be used. This was not a fatal case. When last seen, a year subsequent to the attack, he was a helpless paralytic, with general atrophy of muscles.

The history of a trauma, in a case of convulsions with spastic condition of the facial muscles, is not today presumptive evidence of the presence of tetanus. In a large percentage of cases of frank poliomyelitis there is a clear history of a trauma, frequently accompanied by a solution of continuity. The trauma is most frequently due to a stumble, slip or fall, the result of the inco-ordination which accompanies the onset of this disorder of the cerebrospinal axis.

An adult male 38 years of age, while making stump speeches through a country district where poliomyelitis was epidemic, ran to catch a trolley car, stumbled, fell, and scratched his knee. He was taken ill the following day, treated for lock-jaw and promptly died. The sequence of onset of the acute disease, inco-ordination, fall, and trauma

is indubitably more probable than that tetanus followed so promptly on a slight open wound, and was utterly unresponsive to antitetanic serum, which was administered.

Cases of poliomyelitis occurring within a few weeks of the 4th of July, have been mistakenly attributed to lockjaw resulting from some quite harmless burn from fire or cannon cracker. Every such case, improperly diagnosed, remains a menace to the community unprotected from contagion.

	Tetanus.	Poliomyelitis.
Trauma	Injury not recent; presence of visible wound.	History of trauma probable and recent.
Onset	Seven to fourteen days after injury.	Onset coincidental with trauma or within forty-eight hours.
Course	Early involvement of masseter.	Masseter muscle rarely involved.
Headache	No headache.	Basilar headache intense.
Inferior maxilla	Lower jaw fixed and immovable.	Patient can depress lower maxilla to sternum, though cervical rigidity is marked.

Rabies.—Most children, if not allowed a pet of their own, have established friendly relations with the dog or cat belonging to a neighbor. Much rough and amiable frolicking results, and any observer may assure himself that it is not often the child who is the victim of this rough and tumble sport. An acute and fatal illness, characterized by convulsive seizures with a history of having been scratched or bitten by some household pet, is not necessarily rabies.

On the other hand, there has been an astonishing increase in the newspaper reports of deaths from hydrophobia since poliomyelitis became pandemic in North America. It may be that the curious and unexplained

analogy of these two diseases will not always remain obscure. The physician today should be able to definitely eliminate the possibility of the presence of acute epidemic paralysis before making a diagnosis of rabies.

	Rabies.	Poliomyelitis.
History	Bite or laceration, from rabid dog, cat, fox, wolf, or skunk.	None, or scratch or playful bite from household animal with unimpaired health.
Incidence	Incubation from forty to sixty days.	
Premonitory symptoms	Itching or burning of wound, with renewed inflammation.	Wound healed and invisible.
Onset	Pharyngeal spasm, salivation.	No pharyngeal spasm; no salivation.
Dysphagia	Due to pharyngeal spasm, intractable.	Difficult swallowing due to cervical rigidity and pain. Overcome by use of drinking cup; tonic; unrelaxing.
Spasticity	Paroxysmal.	Tonic and unrelaxing.

DENTITION, AUTOINTOXICATION, PTOMAINE POISONING, ECLAMPSIA, TRICHINIASIS.

Dentition.—The error of an assumed relationship between acute epidemic paralysis, and the period of dentition, apparently dates back to the report of Dr. George Colmer of cases occurring in West Feliciana, La., in the summer of 1841. In this first report of the disease in its epidemic form, Dr. Colmer states that 12 cases were all teething children under 2 years of age. Two errors seem to have taken their origin from this otherwise illuminating report: first, that this epidemic malady was one of infants only, and, second, that it related to the period of dentition.

Dentition is included in the differential diagnosis for the reason that among the laity, and also some of the profession, it has been loosely stated that infantile paralysis was a *complication of teething,* whose results may be and usually are outgrown. A Manhattan father was so informed when his 18-month-old daughter developed a complete paraplegia.

The eruption of the teeth in childhood is usually marked by hypersecretion of the saliva, with drooling, and an enterocolitis with diarrhea. The lowering of the child's resistance would make it more susceptible to exposure from any infection, and therein lies all relationship between teething infants and acute epidemic paralysis. The onset of poliomyelitis is usually accompanied by an obstinate constipation.

Autointoxication.—Intestinal intoxication with retarded elimination and meningism may simulate the onset of poliomyelitis. Elevated temperature, increased pulse rate, basilar headache, vomiting, vertigo, and even a mild delirium or confusion may be all present. The immediate relief given by a thorough flushing of the bowels will serve to differentiate the condition from the disease.

Ptomaine Poisoning.—The furious onset which usually accompanies an attack of poliomyelitis in the adult, may easily be mistaken for a case of ptomaine poisoning from the ingestion of decomposed food. A rapid, irregular, and feeble pulse, projectile vomiting, meteorism, and symptoms of basilar irritation, or stupor, are common to both conditions. In ptomaine poisoning the history will be of an indiscreet diet, of the partaking of some one article of food after which the patient immediately felt unwell, or of some article for which he has always had an individual susceptibility. There may be a history of the simultaneous illness of several members of the same party which had

eaten of the dish, which would clinch the diagnosis. Lavage of the stomach, calomel and a colonic flushing will relieve the vomiting, and the other symptoms will gradually modify, in a case of ptomaine intoxication.

The sudden, forcible, repeated vomiting of poliomyelitis when present, being central in origin, will not yield to stomach lavage, nor will the symptoms of basilar irritation modify immediately under this treatment. There have been undoubted cases during the epidemic disease which were supposed to be due to intestinal irritation, with an extremely severe onset, which cleared up under the treatment outlined, with no remaining paralytic complication, where the true cause of the attack would have been unknown and unsuspected, had not another member of the household subsequently sickened with a typical case of poliomyelitis.

Poliomyelitis.	Ptomaine poisoning.
Epidemic in community.	No epidemic.
History of exposure.	Not exposed.
No dietary indiscretion.	Dietary indiscretion: (*a*) overfeeding; (*b*) decomposed; (*c*) shellfish; (*d*) personal idiosyncrasy.
Vomiting sudden, forcible, repeated, not modified by treatment.	Vomiting distinctly modified by treatment.
Symptoms of meningitis.	Meningism only.
Paralysis.	No paralysis.

Eclampsia.—The onset of poliomyelitis in the pregnant woman closely simulates a uremic attack. The following valuable account of a case, cited by Wickmann, and abstracted by Frost, is given in preference to others known, as the autopsy findings proved the presence of the acute infection:—

H. K., female, aged 27 years, married, taken sick suddenly Aug. 19, 1905, with fever, headache, pains in back; next day vomited so suddenly as to dislocate the jaw; tenderness and stiffness of neck, increasing until head was moderately retracted; violent tonic contraction of the shoulder muscle, throwing the arms up to the head; tonic contractions, flexing elbows, flexing fingers, and adducting thumb; no ocular paralysis; cramps so painful as to require chloroform; evening temperature 99.6° F. Patient fully conscious; opisthotonus. August 21st: temperature 101.8° F.; patient being six months pregnant, eclampsia was suspected and forced delivery was undertaken successfully; cramps continued, extending to legs; inability to swallow and difficulty of speech developed later the same day; condition continued until death at 6 A.M., August 22d. Patient conscious throughout. An autopsy was performed, revealing typical histologic lesions of acute poliomyelitis. The cerebrospinal fluid was found greatly increased in quantity and quite clear.

Here again poliomyelitis has shown its protean character, by closely simulating a condition and disease to which it is unrelated. The points of differentiation in a case of uremic convulsions would be the albumin-loaded urine, possibly diarrhea, sudden amaurosis, and uremic coma. The dyspnea and cyanosis are also much more severe in uremic convulsions, and there may be a history of Bright's disease of the kidney.

Trichiniasis.—Trichiniasis during the period of onset is not so apt to be confused with poliomyelitis as it is at the time, ten days later, of the liberation of the embryos in the invaded muscles.

A poliomyositis is established; the muscles become intensely painful, swollen, and hyperesthetic; an involvement of the diaphragm, which is usual, causes painful and impeded respiration; taken with the history of the attack a few days away, of pain, vomiting and fever, a picture is left on the mental retina which may be closely simulated by the onset of poliomyelitis.

A clear history of eating uncooked pork or sausage must be obtained before the diagnosis can be made. By Herrick's method the *Trichinella spiralis* may be recovered from the circulating blood.

Rachitis.—The characteristic rachitic rosary, the bilateral asymmetry of the paretic legs, and the negative electric reactions will serve to distinguish a case of rickets from the suspicion of the acute infection.

Tuberculosis of Joints With Fixation from Pain.—Professor Eccles, of the medical department of Marquette University, was called in consultation to see a boy of 10 years, with a supposed tuberculosis of hip-joint immobilized by pain. He found a fourth-week case of poliomyelitis, with a clear history of acute onset, occurring while the disease was an epidemic in the State of Wisconsin.

It would seem that the characteristic insidious approach of tuberculosis, with fever, apathy, and emaciation, would be in such marked contrast to the acute onset, sudden paralysis, and rapid atrophy of poliomyelitis that a mistake in diagnosis could not occur.

The affected joint in tuberculosis, in addition to the pain and tenderness, is swollen and perceptibly changed in outline from its fellow. The immobility is that of fixation from pain and is not a paralysis or paresis. The family history may be positive, complicated with deaths from pneumonia or consumption.

The paralytic limb in poliomyelitis is undergoing regeneration, a regression of the paralysis, or rapid atrophy. The affected muscles show the reaction of degeneration, and the entire history is one of an acute disease.

A case of poliomyelitis affecting the left glutei and abdominal muscles strongly resembling tuberculous coxitis is given by Sheffield, of New York:—

J. C., 2 years old, came to the Babies' Hospital Dispensary (Dr. H. E. Hale's division) with the following history: Having been perfectly well up to four days before, he suddenly complained of pain in the legs and seemed to have a slight rise of temperature. The family physician was consulted, but finding no tangible cause for the complaint he administered a laxative and ordered to keep the child in bed for a day or two. The next day the patient was out of bed, but off and on continued to complain of pain, especially in the left leg. It was for this symptom that he was brought under our observation. On examination I found that he was able to make free use of the extremities while in sitting posture and also able to walk, though he did it with reluctance, holding the leg stiff. The child's musculature as a whole was flabby and no perceptible difference could be elicited between the muscles of the different extremities. As pain and a slight limp were at that time the most characteristic symptoms, and as the onset and course of the attack were so exceptionally brief, it required quite a stretch of imagination to pronounce the case as poliomyelitis. Indeed, noting also that there was slight asymmetry between the gluteal regions (which later proved to be atrophy of left gluteal muscles), and on further inquiry having learned that the child had recently sustained a fall, I was rather inclined to the belief that the patient might be suffering from incipient coxitis. I could not help thinking also of rheumatism, pain being the predominating factor. However, all speculative theories soon went to naught. On the second visit, one week later, I found the left gluteal muscles distinctly atrophied and the abdominal muscles equally affected. Moreover, a few days later J. R., a little girl 17 months old, living in the same flat one floor above, was brought to the clinic suffering from complete paralysis of the left leg except the foot, partial paralysis of the right leg, and unilateral paralysis of the abdominal muscles. This child had the attack of poliomyelitis about two weeks before the aforementioned boy, her playmate.

"By reason of their superficial resemblance to hip-disease cases of the above . . . . painful type form a group which is of great importance. Pain, wasting of the buttock muscles, and general weakness are suggestive symptoms; and when to these are added a tubercular family

history, the combination is apt to prove too much for the unwary practitioner, who forthwith diagnosed morbus coxæ. . . . .

"While many authors devote considerable space to the differential diagnosis of morbus coxæ *in so far as it may be mistaken for infantile paralysis*, not one of those I am acquainted with mentions the possibility of the converse error. Yet when the latter mistake is made the consequences are most serious. The child is laid up for weeks and months without the slightest improvement, and I have even come across 1 case where the joint was opened in the confident expectation of finding a tuberculous focus." (Hernaman-Johnson, "On the Occurrence of Pain and Other Sensory Disturbances in the Chronic Stage of Infantile Paralysis.")

It is evident that when a man of Trevelyan's standing is said to have observed acute paralysis following immediately upon such different infections as measles, typhoid fever, and acute rheumatic fever, having obviously been unacquainted with the characteristic measly rash and not infrequent appearance of a typhoid condition and myalgia in acute poliomyelitis, the family physician must constantly have in mind the possibility of any illness with acute onset developing into this disease. The most striking example of mistaken diagnosis we have met with in literature is given by Dr. Lovett, in the account of the cases in the 1909 Massachusetts epidemic, as follows:—

Intussusception of Bowel.—D. G., 2½ years old; August 8th, onset fever, rigidity of spine, retraction of head; vomited; retention of urine fourth day; fifth day removal to hospital and operated for supposed intussusception of bowel. Nothing was found except an excessively distended bladder; on August 18th appeared paralysis of both legs and right arm, thus establishing diagnosis.

Sunstroke and Heat Prostration.—A not uncommon mistaken diagnosis. Presence of epidemic should be considered.

Cerebral Hemorrhage.—The prodromal symptoms of poliomyelitis and the fact that there is seldom any disorientation of time or place in poliomyelitis are sufficient diagnostic points.

Sprained Ankle. Dislocation of the Shoulder-joint.—Acute poliomyelitis with paralysis of the legs in a 4-year-old boy diagnosed sprained ankle, reported by Peabody and Draper. Dislocation of the shoulder-joint not infrequently mistaken diagnosis. Ager, of Brooklyn, called as consultant to the case of a 2-year-old girl, was told that the child had forgotten how to walk.

CHAPTER IX.

Prognosis in Acute Epidemic Poliomyelitis as to Life and Disability.

A GUARDED prognosis should always be given in poliomyelitis. The prognosis is grave while the disease is progressing and the paralysis advancing. The motor symptoms of the preparalytic stage are: tremor, twitching of muscles, jerking of limbs, and spasticity. The paralysis of the diaphragm is usually last; when a patient develops abdominal breathing, and chest muscles are fixed or immovable, give a fatal prognosis, no matter how favorable other conditions may look:—

MORTALITY RATE IN SIX EPIDEMICS OF POLIOMYELITIS.

Reported by	Locality.	Year.	Total cases.	Deaths.	Mortality rate. Per cent.
Harbitz and Scheel	Norway	1905-6	1053	145	13.8
Wickmann	Sweden	1905-6	1025	159	15.0
Hill	Minnesota	1909	283	68	24.0
Lovett	Massachusetts	1909	628	51	8.1
Frost	N. Y. State	1910	227	34	15.0
Bierring	Iowa	1910-11	722	169	23.4
	Totals		3938	626	15.9

The immediate cause of death in poliomyelitis is paralysis of respiration, meningitis, or toxemia, but a contributing cause appears to be strenuous exercise taken after the onset of the disease. Ball, in reporting St. Paul cases, remarked that in the fatal cases the patients had taken a varying degree of exercise after onset. Spiller, in report-

ing a case of poliomyelitis in an adult who walked 8 miles after onset, questions if the disease might not have remained in the abortive (arrested) form, if this exertion had been avoided at onset. (Spiller, "Diagnosis of Poliomyelitis," Penn. Med. Jour., December, 1911.)

The mortality rate of 15 per cent. for all cases rises to 25 per cent. for children less than 1 year of age, for adolescents and for adults. Males, being vastly more liable to the infection than females (Caverly, "Acute Ant. Poliomyelitis in Vermont," Jour. Amer. Med. Assoc., January 4, 1896), show a correspondingly greater death percentage.

The stricter the investigation of cases, the higher the mortality record has proved; thus Hill, discarding every case not presenting an actual paralysis from his record given above, found 68 deaths among 283 cases in Minnesota in 1909, a mortality rate of 24 per cent. We must remember that the inclusion of arrested cases would greatly modify this fatality rate.

The mortality rate varies in wide degree in local outbreaks; Harbitz noted this in speaking of one Norwegian district where 26 cases occurred with 12 deaths, a fatality rate of 48 per cent. This fluctuation was shown in two small outbreaks at seaside resorts in this country; the first one was cited by Morse (Boston Med. and Surg. Jour., January 12, 1911). There were 8 children of about the same age in a small summer colony at the shore. All of them had an acute gastrointestinal disturbance with slight fever lasting from four to five days. One of them was found a week later to have a slight paralysis of both legs and one arm. It is fair to assume the others had poliomyelitis also in an abortive (arrested) form. A doctor's only son, and a girl playmate, died during the acute stage; there were no other cases.

The mortality rate also varies according to age, sex, area, and extent of cord involvement, and, as observed by Shidler, the time of the epidemic in which the case occurs. Shidler noted in the York, Nebraska, epidemic that the last cases had a higher degree of fatality; this may be due to a direct enhancement of the virulence of the infection with the advancing plague; it was seen to occur in the artificial propagation of the disease. The prognosis as to life is more favorable between the ages of 1 and 10 years than in infancy, adolescence, or adult life. Adult males seem to have not more than one chance in two of surviving the attack.

HIGHER MORTALITY IN THE MORE ADVANCED AGES.

Reported by	Age.	Per cent.
Wickmann, Sweden	12 to 32 years.	27.6
Leegaard, Norway	15 to 30 years.	25.8
Fürntratt, Steiermark	Over 15 years.	25.5
Lindner and Mally, Austria	Over 11 years.	50.0
Massachusetts, U. S. A., 1910	Over 10 years.	20.0

(Lovett and Richardson.)

Of 722 cases reported in the State of Iowa in 1910-11 (Bierring), there were 110 cases over 15 years of age. The mortality rate for all, 23.4 per cent., is very high.

Prognosis as to life is also affected by the segment of the cord which is involved, and the extent of the involvement. The higher the lesion the more danger of the involvement of the muscles of respiration, or the vital centers. Prognosis is also grave in direct ratio with the extent of cord involvement. In New York State in 1910 there were 55 cases of paralysis of one leg only and no deaths in that series, while, of 30 cases which presented paralysis of all four extremities, only 12 remained alive; 18 of the cases, or 60 per cent. of the number, died.

In fatal cases there is usually no decline in temperature with the onset of the paralysis.

If the case survives the eighth day the prognosis is hopeful. About 12 per cent. of all cases die during the acute stage, or, to state it somewhat differently, 80 per cent. of all fatal cases die in the first week of the disease.

Sporadic Cases.—It has been stated by several authorities that the mortality rate is much lower in sporadic cases; no evidence having been brought forward, the optimistic hazard remains unproven. If by sporadic it is intended to designate cases which occur singly with no known relationship to other cases of poliomyelitis, whether such relationship has been intelligently sought or not, it is doubtless true that proper investigation would uncover such relationship, and perhaps discover the cause of many mysterious and obscure deaths. It is evident that we must gather much more information about the so-called sporadic form of poliomyelitis before we can more than hazard its mortality rate.

Paralysis, Impending, Progressive, and Regressive.—The motor symptoms of the preparalytic stage of poliomyelitis are: tremor, twitching of muscles, jerking of limbs, and spasticity. During the onset of a case of poliomyelitis it is now possible to prognosticate, by lumbar puncture and the examination of the spinal fluid, whether the illness will eventuate in paralysis. This method was carried to brilliant execution by Drs. Frizzell and Flexner in the case of a Princeton student in 1910. By the use of this method the diagnosis was established, and the probable appearance of the paralysis determined twenty-four hours before onset; a second puncture twenty-four hours after onset of paralysis demonstrated the height of the paralysis passed, and the paralytic stage terminating.

During the acute stage of poliomyelitis the spinal fluid

increases in volume; this is shown by increased pressure when the spinal canal is tapped, for the fluid drops rapidly from the needle or may spurt with a force which projects the stream several inches. Any acceleration of the normal slow drop flow indicates plus pressure. In poliomyelitis

Fig. 66.—Scoliosis following acute poliomyelitis. (N. Y. Hospital for Deformities and Joint Diseases.)

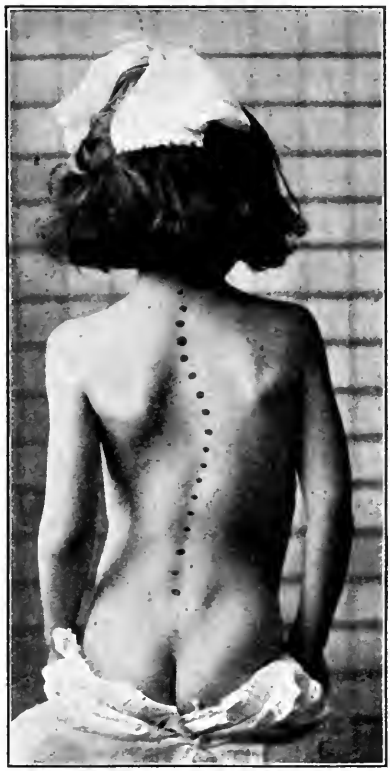


Fig. 67.—Scoliosis following poliomyelitis. (N. Y. Hospital for Deformities and Joint Diseases.)

the spinal fluid is clear during the onset of the disease, becomes slightly opalescent in the preparalytic stage, and again clears as the height of the paralytic stage is reached. This method is the most valuable aid which we possess in the prognosis of both the progressive and regressive stages.

Brorstrom, of Sweden, collected 394 cases of poliomyelitis (1905-6), of which only 79 showed a paralysis of any degree.

Spontaneous recovery frequently occurs in mild cases, and ensues in about 16 per cent. of paralytic cases. In the Massachusetts epidemic of 1909, 25 of 150 paralytic cases made an absolutely complete recovery. These cases were investigated with the utmost thoroughness, and the recovery cases were examined; they were stripped naked and the separate movements of ankle, knee, hip, spine, abdomen, and arms were separately tested; they were found to have made a complete recovery. We are indebted to Dr. Robert Lovett, of the Massachusetts State Board of Health, for this very reassuring analysis. (Bull. Mass. State Board of Health, June, 1910.)

EXTENT OF PARALYSIS IN 25 CASES OF POLIOMYELITIS WHICH SUBSEQUENTLY MADE COMPLETE RECOVERY. (LOVETT.)

One thigh and one leg	4
Both thighs and both legs	8
Both thighs	1
One leg	2
One arm	1
One arm, back, and one leg	1
One leg and back	1
One thigh, leg, arm and forearm	1
One arm and forearm and cervical region	1
Cervical region	4
Indefinite staggering gait (ataxic type)	1
	25

These recovered cases were in no way distinguished from other cases by the character of onset, distribution of paralysis, or tenderness; the paralysis lasted from three days to twelve weeks; the cases ranged in age from 1 to 21 years. The cases all suffered from frank paralysis save

1, which was of the ataxic type. The possibility of such a complete recovery is very reassuring to the parents, although a prognosis in this disease must at all times be guarded.

Although the symptoms of onset are not dependable in prognostication, it has been frequently observed that cases which begin with a diarrhea are usually mild in type, and the paralysis usually transitory; while cases presenting the profound constipation are more serious in degree. When the decline in temperature precedes the paralysis more than forty-eight hours, the paralysis is usually of a mild type.

DISTRIBUTION OF PARALYSIS.

	Mass., 1909 (Lovett).	New York State, 1910 (Frost).
One leg only	192	55
Both legs only	151	65
One arm only	32	16
Both arms only	11	8
Arm and leg	74	28
Both legs and one arm	38	12
One leg and both arms	6	2
Both legs and both arms	82	30
Back	83	34
Abdomen	37	20
Face	34	13
	740	283

Residual Paralysis.—Spontaneous recovery from the paralysis may be rapid for several weeks, when no farther advance is observed. The complete recovery of one extremity, while its fellow is left with a serious residual palsy, is most often seen. The percentage of cases left with a residual palsy has been variously estimated. In an editorial in Pediatrics (August, 1910), it is given as 80 per cent. This estimate is somewhat higher than was found by Hill

in his study of 283 paralytic cases. Of the 283, a good recovery was made by 15 per cent., while 24 per cent. died. There remained then 60 per cent. more or less crippled by the attack; but as Hill in this study excluded all cases of the arrested, ataxic, neuritic, or meningitic type which did not show a distinct paralysis, it becomes evident the in-

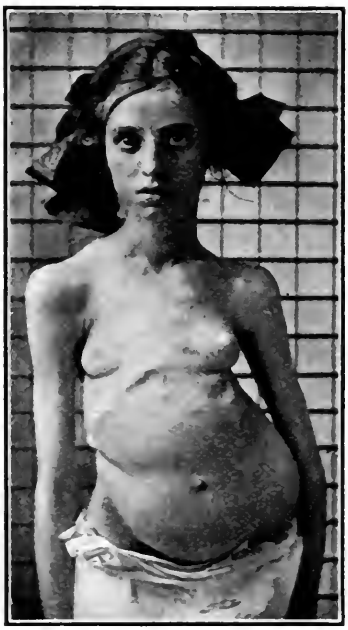


Fig. 68.—Paralysis, atrophy, contractures and deformity eighteen months after attack of poliomyelitis, in a girl 11 years of age. (N. Y. Hospital for Deformities and Joint Diseases.)

clusion of such cases would have lowered the percentage of cases of persistent paralysis and atrophy to somewhat below 60.

Cortical involvement may occasion convulsions, hemiplegia, mental deficiency, and epilepsy. The mental deficiency may be any one of the three grades: Idiots, those children who never develop beyond the mental age of 3

years; imbeciles, individuals retarded mentally to the age of 7 years; morons, those high-grade fools that remain at the mental age of 12 years.

Fig. 69.—Posterior view of Fig. 68.

Fig. 70.—Lateral view of Fig. 68.

ARREST OF GROWTH OF LONG BONES AND AMOUNT OF SHORTENING.

Arrest of growth is proportionate to growth rate, and the younger the child affected the greater the possible shortening. The degree of shortening usually bears some proportion to the degree of severity of the paralysis, but this is not constant and not at all dependable for prognostic purposes. An extremity with a light paralytic attack which occurred in early infancy may have almost unimpaired function and yet present a shortening of four or more inches. In an extremity with an extensive flaccid

paralysis there may be an actual shortening of less than an inch, and very occasionally cases have been noted of actual lengthening. Trophic disturbance of bone development is usually associated directly with muscle involvement, as the tibia and fibula are most apt to show shortening when the muscles below the knee are paralyzed, while the involvement of the muscles of the thigh usually accompanies shortening of the femur.

ATROPHY; HYPERTROPHY; TIME OF RECOVERY.

In Wood's study of 57 cases reported recovered in the Massachusetts epidemic of 234 cases in 1907, it was found that there were three classes of recovery: (1) complete recovery without atrophy; (2) recovery with complete function, but with some atrophy; (3) recovery with some hypertrophy of the affected limbs.

The table shows the proportion of each of these:—

		Per cent.
Complete recovery without atrophy	16	28.1
Functional recovery with atrophy	21	36.8
Recovery with some hypertrophy	3	5.3
Recovery, presence or absence of atrophy unknown	17	29.8

(Wood.)

Leaving out the last group, and averaging those in which the presence or absence of atrophy is known, shows that for every 4 recoveries without atrophy there were 5 with atrophy. When atrophy was present the maximum amount noted was as follows:—

	Inches.
Calf	1¾
Thigh	1¼
Arm	½
Forearm	¾

In most cases the atrophy was much less than this, a difference of one-eighth of an inch being counted as atrophy, but in all such cases examined function of every muscle was perfect. Three cases had only one-eighth inch atrophy of one limb; 2 cases only one-fourth inch of one limb; 5 cases only one-eighth inch of one limb and one-fourth inch of another; the other 11 cases had more than one-fourth inch atrophy. The amounts of hypertrophy recorded were in 2 instances one-fourth inch and in 1 three-eighths inch, all of the calf.

TIME OF RECOVERY.

	Cases.
1 week or less	2
1 week to 1 month	8
1 to 2 months	8
2 to 3 months	5
3 months to 6 months	10
6 months to 12 months	9
1 to 2 years	5
2 to 3 years	5
No data	5
Total	57

(Wood.)

To illustrate late recovery, and recovery from severe attack, 2 cases were especially notable:—

W. H. and J. H., brothers, aged 11 and 13, patients of Dr. J. C. Hubbard, of Holyoke, were attacked Oct. 12 and 13, 1907, respectively.

The duration of the acute attack was three weeks in each case, that of W. H. being moderate and that of J. H. severe. The latter had a temperature of 103° F. for a week, vomiting for several days and severe coma for two weeks, with marked retraction of head. The distribution of his paralysis at its worst was both thighs, left leg, left arm and left forearm, and lower back. The arm and back recovered in one month.

The distribution of W. H.'s paralysis was left thigh and left leg.

The treatment was the same in both cases and consisted of massage and tabetic exercises begun in November, 1907, after the acute onset.

Both patients were in bed three weeks and had to be carried for two months and then walked with a limp, the lameness being very marked in the case of J. H. during the first year. Massage was given every night for two years. The greatest gain occurred during the second year and practical recovery occurred during the third year.

An examination in May, 1911, showed no paralysis in either case. W. H. had one-eighth inch atrophy of left calf and one-half inch of left thigh. J. H. had one-eighth inch atrophy of right calf and no atrophy of the thigh or arm.

The following conclusions seem justified:—

In anterior poliomyelitis complete recovery or functional recovery occurs in something over 25 per cent. of cases examined at the end of four years. Atrophy may exist without impairment of function. In about half of the recovered cases, the onset was mild. The distribution of the paralysis in such recovered cases was not essentially different from that in cases which do not recover. Recovery in many instances required months, and in several cases from one to three years. (Wood, Bulletin Massachusetts State Board of Health, 1912.)

Summary.

A. The motor symptoms of the preparalytic stage of poliomyelitis are:—

Tremor.
Twitching of muscles.
Jerking of limbs.
Spasticity.

B. Lumbar puncture and examination of spinal fluid may determine:—

Paralysis (*a*) impending.
(*b*) progressive.
(*c*) regressive.

C. The prognosis of poliomyelitis is hopeful when:—

1. The patient is more than 1 and less than 10 years of age.
2. With mild onset (not dependable).
3. With diarrhea at onset.
4. With decline of fever by crisis preceding paralysis.
5. When decline of fever precedes onset of paralysis by more than forty-eight hours, usually very mild type.
6. With paralysis of one lower extremity.
7. When paralysis is regressive in type.
8. If patient survives eighth day.
9. With absolute rest in bed from onset.

D. The prognosis in poliomyelitis is grave when:—

1. The patient is less than 1 year of age, an adolescent, or an adult.
2. When onset is characterized by severe symptoms (not dependable).
3. With suppressed eliminations.
4. With strenuous exercise or exposure to heat after onset.
5. With no decline in temperature as paralysis supervenes.
6. While paralysis is advancing.
7. With paralysis of all extremities.
8. With paralysis of upper segments.
9. With paralysis of diaphragm, intercostals, or muscles of pharynx.
10. Character of epidemic (virulent).
11. Time in epidemic (late).

CHAPTER X.

Prophylaxis of and Immunity from Poliomyelitis.

COMMUNAL; INDIVIDUAL; PHYSICIAN.

THE experimental transmission of acute poliomyelitis has been effected *only by inoculation,* by (*a*) the injection of a healthy animal with poliomyelitic tissues; (*b*) the bite of biting stable-flies previously allowed to feed on poliomyelitic animals; (*c*) the injection of healthy animals with the comminuted bodies of bedbugs previously fed on poliomyelitic animals. The spontaneous transmission of acute poliomyelitis has been known to occur from inoculation of man by a paralytic dog. (Langhorst.)

There is no proof that spontaneous transmission of acute poliomyelitis, without an inoculation wound, can take place. There is no proof that contact contagion takes place. Spontaneous development of the disease among laboratory animals is unknown. The toxic buccal secretions (saliva) of a case induce the disease only when injected into the healthy animal through an inoculation wound. Fomites, said to carry contagion, have been shown infective only by injecting water in which they were laved into an inoculation wound.

COMMUNAL PROPHYLAXIS.

In view of the foregoing, rational communal prophylaxis against epidemic poliomyelitis must be wider in scope than the hitherto tenuous advice to secure street sprinkling for the city, and nasal antisepsis for the citizen. Adequate

measures must be taken to secure the citizens immunity from transmission of the disease from

(1) the human case;

(2) the paralytic animal, dog, horse, hog, sheep, rabbit, pigeon, barnyard fowl, cat;

(3) blood-sucking insect, parasite of animal and man,

(*a*) the biting stable-fly, *Stomoxys calcitrans;*

(*b*) the bedbug, *Cimex lectularius;*

(*c*) all other blood-sucking insects: mosquito, louse, flea, tick, buffalo gnat, midge, sand-fly, and the itch;

(4) contact contagion from common carriers, such as roller towel, public drinking-cup, etc. (until theory of contact contagion is absolutely proven or disallowed).

In addition it is advisable for each community to appoint a special physician, salaried, as advisory consultant, epidemiologist, and delegate to an international clearing house which will give warning of the approach of the epidemic disease, with a special council to take measures to check its advance.

1. THE HUMAN CASE AND COMMUNAL PROPHYLAXIS.

A legal statute making epidemic poliomyelitis a disease subject to public control is the first requirement in communal prophylaxis. The London Board of Health requires notification of this disease with a fine of $10 for every failure to report a case. It is doubtless true that such a law and such a forfeit for failure to comply with the law, if enforced, would bring knowledge of thousands of unreported cases occurring each season in the various States, each unrecognized case a communal menace. The enforcement of the fine might result in more careful diagnoses. Poliomyelitis was, until recently, not a reportable

disease in the State of Missouri; a communication from Dr. B. S. Veeder, of the Children's Hospital of Washington University, St. Louis, during the fall of 1912, states that the writer is not sure that an ordinance making poliomyelitis reportable would be effective, "as cases come into the hospital under the diagnoses of rheumatism, neuritis, and diphtheria of the stomach" (!). The strict enforcement of the law requiring that each case be reported to the proper authorities has, in Sweden, brought the exact knowledge of 3800 cases occurring in the summer of 1911, and 1800 cases up to August, 1912; so that the government at least knows what it is up against and can take measures accordingly.

A. Classification of poliomyelitis with epidemic diseases under public control.

B. Enforcement of notification by penalty of at least $10.

C. Report to include précis of case, with environment and probable source of infection.

D. Isolation of case for four weeks from human companionship, save that of necessary attendants.

E. Isolation of case from all blood-sucking insects; absolutely maintained by screening, and partial fumigation.

F. No domestic pets permitted in or about chamber.

G. Disinfection of all dishes, linen, secretions, and excretions before removal from sick chamber.

H. Quarantine of all who were in close association with case for a period of ten days preceding onset of disease, such quarantine to be maintained for ten days.

I. Isolation of all cases developing a coincidental febrile condition, malaise, cervical rigidity, or motor weakness, for a period of four weeks, the same precautions regarding blood-sucking insects to be enforced.

J. Placarding of house with the name of the disease given plainly.

K. Members of family excluded from school. Breadwinners to take their meals elsewhere and use separate toilets until after final fumigation.

L. Final fumigation of premises with sulphur gas under pressure or an equally valuable method. Formaldehyde fumigation for bedbugs was pronounced worthless by Verjbitski after a complete investigation.

COMMUNAL PROPHYLAXIS AND THE LOCAL HEALTH BOARD.

During the Springfield, Mass., epidemic of 1911, the following were used by the Springfield Board of Health, a copy of the circular sent to doctors being given below:—

HEALTH DEPARTMENT, SPRINGFIELD, MASS.

ANTERIOR POLIOMYELITIS.

The State Board of Health has declared anterior poliomyelitis to be a disease dangerous to the public health, and as such must be immediately reported to the local board of health. Failure to comply with this statute involves liability to a fine of $50 for each offence.

From all information obtainable of epidemics of this disease in other places, as well as a study of the epidemic now in progress in this city, the disease is without doubt of an infectious and contagious nature. The causative agent is not known, but the mortality and subsequent paralyses and deformities, which are only too much in evidence, place anterior poliomyelitis amongst the diseases most dangerous to life and future usefulness.

The board of health has therefore established a strict isolation period for this disease of four weeks from date of notification, with disinfection at termination of the period. No public funerals will be allowed of patients dying from this disease.

It is only, however, through the earnest and active co-operation of the medical profession, who come in personal contact with the disease, that definite results can be obtained by the board of health in checking the spread of the epidemic.

Physicians are, therefore, urged to use every precaution in preventing its spread which is now in use in the management of other contagious diseases, and to explain very carefully to the family the dangers arising not only to themselves, but to others. from a disregard of the regulations of the board of health. If this line of action is pursued by the medical profession, it is believed

that as good results will be obtained as to undertake the establishment of an absolute quarantine, with all its discomforts, hardships, and consequent tendency on the part of the public to conceal the existence of the disease.

If physicians will advise all their families, as a matter of self-protection, to at once establish a voluntary isolation for themselves, the best quarantine possible will then be produced. Children should be kept at home and not allowed to attend picnics, excursions, Sunday school, theaters, playgrounds or other places where children come together in numbers.

July 27, 1910.

HEALTH DEPARTMENT, SPRINGFIELD, MASS.

ANTERIOR POLIOMYELITIS (INFANTILE PARALYSIS).

This disease is contagious, and is followed in a great many cases by paralysis of arms, legs, or other parts of the body. Deformities with inability to use the arm or leg are liable to be permanent.

It attacks young children chiefly, but adults are by no means exempt. It is therefore of the utmost importance that you use every means possible in preventing other children from contracting this disease.

No one will be allowed to see the patient except the necessary attendants and the physician. The patient must be cared for in a room isolated from the other members of the family during the entire period of isolation. Mild cases which are only sick for a few days, and do not show any definite paralysis, are just as liable to convey the infection as the more serious cases, and must be isolated for the full period. Every case should remain under the care of your physician during the entire isolation period, which is four weeks from the date of notification sent to the board of health. Disinfection will then follow and the card be removed.

If the regulations of the board of health are not observed and the patient for any reason is not or can not be properly isolated, the board may cause such patient to be removed to the isolation hospital. The importance of obeying these instructions cannot be overestimated, as a large percentage of children who contract this disease will be deformed or crippled for life with consequent inability to work or provide for themselves.

It is a duty you owe to your children and those of your neigh-

bors to exercise every precaution in preventing the continuance and spread of this disease, and unless you exert yourself in this matter, an absolute quarantine of all cases of anterior poliomyelitis will be declared by the board of health, which will inflict great hardship and distress not only upon yourself, but upon the entire city.

Human life is the most valuable asset we possess, and it must be protected at any cost.

Special emphasis should be laid on the suggestion that all children's gatherings be discountenanced by the local authorities, such as, for example, picnics, excursions, public playgrounds, theaters, shows, Sunday schools, etc.

That the clothes and linen used by the household be disinfected and excluded from the public laundry.

That the school committee be advised to extend the vacation a fortnight, thus postponing the reopening of schools until the 19th of September (the school committee were unanimous in their vote on this question, granting the extension of the vacation, and further agreed that cases of epidemic poliomyelitis, that had occurred in Springfield that summer, be excluded from the public schools for at least two or three weeks after the isolation order was raised), and that a special placard be posted on all the houses in a conspicuous place. The following is the form used in Springfield:—

KEEP OUT.

Anterior Poliomyelitis.

All persons, not inmates, are forbidden to enter this building.

BY ORDER OF THE BOARD OF HEALTH.

Any person removing this card without authority is liable to a fine of from $10 to $100.

2. THE PARALYTIC ANIMAL AND COMMUNAL PROPHYLAXIS.

There is no class of domestic animals nor fowls which has wholly escaped attack from epidemic paralysis coin-

cidental with acute poliomyelitis in the human family. In some communities it has been noted that colts and horses were most frequently affected, and this is doubtless due to the fact that sickness of an animal of the monetary value of the horse will certainly be noted. Frequently dogs have become paralytic, and Langhorst, of Illinois, observed 2 cases of acute poliomyelitis in the human develop from canine infection. A notable case of association of epidemic paralysis of animal and man was reported by Ely, of Iowa, during the Iowa epidemic of 1911. A Boone County, Iowa, farmer, observing that a number of chickens became paralytic, snapped off their heads and threw them into the hogpen; later in the summer a hog developed paralysis of the hind quarters, and somewhat later the farmer's child developed an attack of acute poliomyelitis, which was attended by Dr. Ely. Another paralyzed fowl, being observed on the farm at this time, was submitted to the pathologist at Drake University, who found in the cord of the chicken the histologic picture of acute poliomyelitis in man.

Since it is positively known that the biting stable-fly is the agent of transmission of acute poliomyelitis from animal to man, communal protection now demands the death of all small and inexpensive domestic animals which show symptoms of paralytic disease, and the incineration of their bodies.

In the case of more valuable animals, the horse, blooded cattle, sheep, pedigreed cats and dogs, it would be sufficient to enforce the compulsory screening with wire mesh of the quarters in which the animals are confined, with a sanitary maintenance, which inhibited the breeding of flies, of all environment. The owner of the animal should be fully informed that by so doing he is maintaining a possible menace to his remaining stock, as well as the members

of the household or servant staff. This is a large and as yet uninvestigated subject. Our present knowledge of disease carriers would suggest to the thoughtful that the wry-necked colt and the poke-necked horse may remain a carrier of the organism of poliomyelitis for life. The community which demands the destruction and incineration of all paralytic animals, and at the same time gives attention to the insect problem, will thus assure the best prophylaxis to its citizens.

The Dog.—There are two measures which should be taken by every community and made permanent. Dogs should not be allowed to roam at large. All dogs on the street or public highway should be muzzled. There is abundant evidence that poliomyelitis and rabies are so closely allied as to be for all practical purposes classified as identical diseases. The sick dog while in the febrile or furious stage scatters infection by biting the men and animals with which he comes in contact. During this period and also in the paralytic stage he may, through the agency of biting flies, remain a source of infection of men and animals. As muzzling, while needed, is only a partial protection, the sick dog, if too valuable to be shot, should be kept in a pen screened with a wire mesh which will exclude all biting insects, his pen to be thereafter burned or disinfected to destroy all fleas.

There are two ways of safely disposing of the body of the paralytic animal. It may be incinerated with fire, or buried in quicklime. Never under any circumstances should the paralytic animal be fed to other stock. Burial is equally dangerous. Proescher, of Pittsburg, relates that virulent rabic virus was found present in the body of a dog which had been buried for eight months. Pasteur found that earthworms brought anthrax germs to the surface from the bodies of sheep which died of that dis-

ease and had been buried in somewhat shallow graves. The physician and officer of the health board should advertise these measures through the local press and with circulars.

It has been definitely ascertained that animals contract epidemic poliomyelitis, a disease whose symptoms are identical with the so-called rabies. There is no man today who can state with authority that a paralytic animal has contracted rabies and has not contracted acute poliomyelitis.

3. BLOOD-SUCKING INSECTS PARASITIC TO MAN. PROPHYLAXIS.

(a) The Stable-fly. The Barn-fly. The Rain-fly (Stomoxys calcitrans).

The stable-fly is best known by the fact that, while it closely resembles the common house-fly in appearance, it bites, which the house-fly provided only with a sucking beak cannot do. In the Mid-West it is best known, by the name of its characteristic attribute, as the biting fly. A frequenter of stables, it is fairly common in insanitary homes, and as it always seeks shelter before a storm the proverb, "Flies begin to bite before a rain," became traditional. From this fact the insect in England is known as the rain-fly.

The biting stable-fly transmits the micro-organism of infantile paralysis directly from the blood of an infected case to the next animal or man which it attacks. This fly was found on the premises of a majority of cases of poliomyelitis investigated by Sheppard during the Massachusetts epidemic of 1911. It is the writer's belief that the stable-fly is the common agent in transmission of this disease from animal to man, and that its agency between man and man is less immediately dangerous. The menace

of its presence is such, however, that its destruction must be achieved by the sanitary community.

Methods for Destruction of the Biting Stable-fly and Other Flies.—Animal excrement and especially horse-droppings maintain the most fertile breeding ground for flies. Garbage which is left exposed about houses, hotels, butcher shops, and slaughter houses will be found alive with maggots. There is a fly in the Mid-West which maintains vast breeding places on the borderline of fields near water, where millions of flies may be seen breeding. The breeding places of flies must be found and rendered sterile. The most common breeding place is the untended stable. The floor of a horse-stall which is not cleaned once daily will be found, during warm weather, to be alive with maggots under the bedding..

A valuable method of immediate sterilization of stable manure, as used on a model farm, is here given:—

> Mix equal quantities of acid phosphate and German kainit, and throw a shovelful in the gutter back of each horse twice daily. The mixture absorbs the liquid manure, prevents manure from burning, adds greatly to its plant-food value, and prevents the breeding of flies in the manure. *Caution:* Do not allow much of the material on the platform where the animals stand, as it might make their feet sore if they stood constantly on it.

In addition to this precaution, the manure should be kept in a closed container which is provided with a wire-trapped airhole to catch and retain any flies which may breed in its contents.

The tight container with wire fly-trap at top should be made compulsory for domestic garbage, hotel use, and the ground bone, offal, etc., of butcher shops.

Each community will have to adjust its own problems in this particular and will find sufficient work in cleaning up the detestable fly-breeding conditions maintained about

country slaughter houses, canneries, bakeries, etc. The communities which first appreciate the value and enlist the services of the women home-makers, whose interests place them above the graft of inspectors appointed by the local politicians, will first attain a sanitary condition sufficiently excellent to halt the spread of any epidemic whose germ is blood-borne.

The fly-killing fungus which has been grown in pure culture by Hesse, Park and Copeman, of England, has been found to be severely pathogenic to house-flies, and it is not impossible that it may prove to be destructive to other flies. The fungus is under consideration at the United States Entomological Bureau. This fungus is said to be so fatal to flies that if one fly is inoculated and set free, an epidemic spreads among flies and they drop by thousands, to be gathered up and burned. The fly is inoculated by scratching it lightly with a needle which has been touched to a pure culture of the fungus.

Tanglefoot, traps, and fumigation are as effective with the *biting fly which gains entrance to the house* as with the house-fly. A new method of poisoning them could possibly be devised with fresh bananas, as they will feed on this fruit, but are said to not frequent nor be attracted by watery solutions.

(b) The Bedbug. The Indo-European Bedbug (Cimex Lectularius).

"Creeping things from which come obscure diseases." Columella, 62 years B. C.

The bedbug is probably the common carrier of acute poliomyelitis from man to man, and its extermination is the goal to be aimed for. The bedbug must be excluded by destruction from all dwellings, business districts, public halls and utilities, hospitals and churches, free dispensaries

and night shelters, rolling stock, and passenger ships. An annual spring extermination of the bedbug will result as well in the destruction of all hibernating house-flies and house-mosquitoes, cockroaches, ants, and house-centipedes. An annual renovation to be effective must include not the bed-chambers of a house only, but every room in the house; not a single flat in an apartment house, but every floor, including the basement; not one house in a tenement row, but the entire block.

Methods for Destruction of the Bedbug.—The methods to be employed are (1) general, including fumigation by hydrocyanic acid gas or brimstone, or destruction by fire; (2) local, including the application of various volatile or other poisons, or of boiling water.

Hydrocyanic acid gas method: The immediate and complete sterilization of a building and all its contents, with the total destruction of all germs, parasites, and vermin on the premises, can be accomplished by fumigation with hydrocyanic acid gas. The gas penetrates every aperture, and leaves behind it the death of every organism; the dust will be undisturbed but innocuous; no cockroach or water-bug will be seen about the plumbing; no bedbug, moth, mosquito, or house-fly will emerge from any crevice or drapery. Fumigation by this method is so exact that it should be in common use at least once a year; its immediate effectiveness recommends it for use in all aggravated and intractably insect-ridden houses. The method, however, is both expensive for individual use, and dangerous when employed unintelligently. Individual experimental use of the hydrocyanic acid gas method is not advocated. It should be a part of the equipment of the municipality which is placed at the call of the individual citizen, as is fire protection, and should be compulsory in all buildings enumerated above during the months of spring. Annual com-

pulsory municipal fumigation can be accomplished by municipal ownership of equipment and sufficient accessory apparatus in the way of tents, etc., to house for twenty-four hours the inhabitants of one city block, notification to be given one week in advance. The hydrocyanic acid gas method of fumigation of houses is given in detail by L. O. Howard, Chief of the United States Bureau of Entomology, in Circular Bulletin 46, revised edition. In response to a letter of inquiry regarding the use of the hydrocyanic gas in tenements, Dr. Howard replied: "In order to be effective and safe in East Side tenements, fumigation with hydrocyanic gas should only be carried on in the central one of three vacated buildings. I doubt very much whether one could get the people out of an entire block in that congested quarter, or even out of one building." Yet it is in the slums of our great cities in which pestilence breeds and from which it spreads. It is in these slums that bedbugs abound.

Sulphur fumigation: It is sometimes very desirable in the eradication of parasites to fumigate a room at a time, or to fumigate several rooms successively. Summer camps and seaside cottages may be infected with bugs and should be fumigated before occupancy. A room or a relatively small house can be satisfactorily treated by sulphur fumigation. Sulphur will bleach books or draperies and all metal will tarnish. The sulphur candle is convenient for use. Use 1 candle for a closet of ordinary size and for a room which can be sealed use 1 pound of sulphur candles to each 1000 cubic feet of air space. It will not be necessary to seal a vacant frame building, but it will be necessary to increase the amount of sulphur to 2 pounds for each 1000 cubic feet of air space and close the building for treatment at least twenty-four hours. The sulphur should be placed in a good-sized receptacle such as a coal-hod,

which should be placed in a pan of water, so that there may be no overflow of the burning mass to start a conflagration. Close all windows, registers, ventilators, and large apertures, but do not paste up broad cracks with strips of paper, as by that means may be preserved alive the vermin you wish to destroy. Ascertain that the sulphur is well fired and burning, and close up the room or cottage for twenty-four hours. The premises should be well aired for a day succeeding the fumigation before occupancy.

Destruction by fire: When small structures of moderate value, such as chicken-coops, outside closets, and summer camps, have become infested with bedbugs, the best prophylaxis is incineration. A great deal of old household articles stowed about the country house should help to feed the fire. Old and unused bedsteads of wood, folding beds, picture-frames, and trunks are especially liable to be infested. The rubbish in attics becomes infested with bedbugs brought to the chimneys by swallows and bats, and these parasites can no longer be considered harmless. Fire can be used effectively in freeing an iron bedstead which is thoroughly infested with bugs; bugs sometimes take complete possession of a neglected bed in an institution, and will be found to have invaded any joint or crevice in the gas-pipe of which it is often constructed. The bed must be removed to a concrete floor, or out of doors; alcohol, poured about all crevices and joints and immediately fired, will destroy every bug and all the eggs; the wire springs can be fired by passing a burning newspaper torch below them.

Local application: The application of corrosive sublimate, the petroleum oils, and the various insecticides is not advocated for the reason that, while any of these agents will kill a certain number of bugs, they will at the same time drive others to a different bed or another room,

and the fight must be soon renewed; also, the treatment of a few pieces of furniture or a portion of a house is mere temporizing, for owing to the secretive habits of the bedbug a small number only will be destroyed. Water, poured from the teakettle in which it is still boiling, will kill all bugs and eggs on the area to which it is applied. Quicksilver, beaten to an emulsion with egg albumin, is very destructive, as the bedbugs will feed on the fresh albumin and die.

As it is possible that poliomyelitis may be transmitted by other biting insects parasitic to man, the most practical insecticides and methods of prophylaxis for the flea and mosquito are given:—

Flea.—Usually brought to house by dog or cat; eggs deposited on rug or floor of animal's bed. Burn the bedding. Cover the floor with loose crystals of naphthalin, allow them to remain for twenty-four hours, then sweep up; may be used again. When fleas infest a living-room scatter the naphthalin over floor, close for twenty-four hours, and sweep. Five pounds of naphthalin are sufficient for a medium-sized room, and may be used repeatedly. (Doane.)

Mosquito.—The absolute draining of all standing water will largely prevent the breeding of mosquitoes. Screening, fumigation, the burning of pyrethrum powder, and the kerosene cup form secondary relief.

"The complete destruction of the blood-sucking insect is the goal. Though it may never be reached, yet the reduction in numbers and the gradual elimination from definite areas will proportionately reduce the dangers. This warfare demands an accurate knowledge of every detail of the life history of these insects, the appearance presented by their stages of development, their breeding places, their food, the length of the period of development.

and the resistance of the larval and other stages to various destructive agencies. Often a very weak and a very inconspicuous link in the chain of development can be easily attacked, and life thereby destroyed, or breeding rendered impossible." (Theobald Smith.)

4. CONTACT CONTAGION FROM COMMON CARRIERS.

We know that the organism of poliomyelitis is viable in the buccal secretions (saliva) of a case at least during the acute stage. It has not been demonstrated that these secretions render the disease contagious, but it cannot be gainsaid that an infection from these secretions might be contracted through the intermediary of household or public linen or the public drinking-cup.

Federal law has now abolished the public drinking-vessel; in many States the common towel has been abolished. Paper cups, paper towels, paper handkerchiefs, and paper napkins should be adopted and advocated by every sanitarian.

FUMIGATION OF THE COMMON CARRIERS OF MODERN TRANSPORTATION.

"An entirely healthy Vienna woman of 25 years spent five days in Berlin with her parents. A strong draft blew through the sleeper on her way to Berlin. On her return she developed an acute ascending poliomyelitis, dying in stupor on the ninth day." (Blum, Wiener klinische Wochenschrift, Sept. 5, 1912.)

One of my husband's men, returning from Bridgeport to New York a summer night in 1912, by the way of a Sound steamer, found the bedbugs so numerous in the men's ordinary that sleep was not possible; on complaining to the clerk he was informed that the only thing which could be done was to take a stateroom.

There are cheap, effective, and well-known methods for securing the destruction of blood-sucking insects which congregate about the seats and sleeping berths of common

carriers. The fumes of burning sulphur are recognized as a standard remedy for the fumigation of barracks, and under the name of Clayton gas, introduced under pressure, sulphur dioxide is now extensively used for the fumigation of ships and their cargoes.

The Engineering News of August 29, 1912, printed the picture of a tank 16.4 feet wide and 75.5 feet long for disinfecting passenger coaches, which is used on the Prussian state railways. A coach is rolled in on rails, the door is closed and hermetically sealed, a partial vacuum is produced, and steam pipes boiling water at 120° F. complete the process of killing all vermin. They cannot endure the temperature and reduced air-pressure combined. A germicide in vapor form is then introduced to destroy all bacteria.

Fumigation of sleeping cars every two months, with airing and cleansing of all berth clothing at the end of each trip, were measures proposed for State legislation at the meeting of the Northwestern Railroad Sanitation Conference held in St. Paul, Dec. 5, 1912. Measures such as these should receive the support of every physician, and such support is most effectively given through the medium of the county and State medical societies.

INDIVIDUAL PROPHYLAXIS.

Two frequent questions asked the doctor during the appearance of poliomyelitis in a community are:—

What can I give the children so they won't take it?

What can I do to keep from getting it?

In Wisconsin in 1908 I advocated removal to an uninvaded district until the height of the epidemic had passed; and it is amazing how little we can add to this suggestion today, although we realize more clearly the disadvantage to the uninvaded community of such visitations from an infected district.

Environment.—Close association with a recent case of poliomyelitis is now considered undesirable; such cases should be provided with a separate chamber and bed. Association with sick animals is to be avoided at all times. Domestic pets when ill should be isolated as certainly as sick children until the character of the illness is known. There has been a constant association in the recent epidemics of coincidental epidemic paralysis of almost every domestic animal. Cats, dogs, hogs, chickens, etc., paralyzed after acute illness, should be killed and their carcasses burned; but it may be easier to remove one's habitation than to insist on the destruction of another's property. The individual who is not a householder should see that his immediate environment is sanitary, or remove to other lodgings. A chamber should have bare floors and little drapery, should be mopped once a week, and be fumigated with sulphur if there is a possibility of vermin. Schoolteachers should ascertain that the schoolrooms in which the day is passed are sterile and free from dust, even though an appeal must be taken to the schoolboard to secure it.

Personal cleanliness is also a factor in prophylaxis. Public linen and public drinking-cups are to be avoided. This applies to the cup on the highway; also applies to the tumbler of water passed about among a group of friends at a social game of cards or elsewhere, for we cannot ignore the fact that poliomyelitis as well as many other diseases may be transmitted from the secretions of the nose and throat.

Immunity and Immunization.—A relative spontaneous immunity to poliomyelitis seems to be possessed by some fortunate individuals; it is demonstrated not so much by the members of a family who wholly escape as by the large percentage of cases of the arrested type: people who have

taken the infection, but seem to be able to at once shake it off. A free, natural elimination would seem to be the factor in this resistance, and the theory is supported by experimental proof,—the artificial production of the disease in monkeys by way of the digestive tube was unsuccessful until the digestion was inhibited by opium.

Artificial immunity to poliomyelitis may perhaps be acquired by the most rigorous attention to elimination. Calomel every second or third week-end, cascara sagrada daily at bedtime, and copious drinking of pure water at and between meals are excellent measures to advocate to the sedentary American. Better than the advocacy of any particular measure is to convince an individual of the fecal residue forming an excellent culture media for germs which remain in his or her digestive tube after the "regular daily movement."

Serologic immunization has been attained for the ape, but no serum immunity for man has yet been perfected.

Medication.—The most important medication in poliomyelitis, the use of eliminants, has just been discussed.

Internal Antiseptics.—Hexamethylaminetetramine has been advocated for use in the prophylactic treatment of infantile paralysis, as it has been recovered from the spinal fluid one-half hour after ingestion. Its usefulness is questioned by others, while its known action as an irritant to the bladder suggests caution in prescribing it to children. A reliable intestinal antiseptic, which is recommended after years of use in general practice, is bismuth betanaphthol. I would suggest that, at least in childhood, this drug be substituted for the preparations of formaldehyde.

Disinfection of Nose and Throat.—The individual who does not make use of peroxide of hydrogen as a dentifrice is advised to add it to his toilet table. Peroxide is the most advocated of all disinfectants in the prophylaxis of polio-

myelitis, as a 1 per cent. solution is said to destroy the virus. It is an excellent dentifrice, and its use as such insures a constant rather than a spasmodic cleansing of the mouth and throat. It is also an excellent gargle. I do not advocate its use as a nasal douche, as it oxidizes tissue so rapidly as to unpleasantly clog the nares. The nasopharynx may be cleansed daily with the following antiseptic spray:—

℞ Thymolis ..... gr. xv.
Sodii boratis,
Sodii bicarb. .....āā gr. lx.
Fl. ex. pinus Canad. ..... ℥ss.
Glycerini ..... ℥j.
Aquæ .....q. s. ad ℥iv.

For atomizer use 1 dram to 1 ounce of warm water.

NASAL ANTISEPTICS. A WARNING.

The appended article by Dr. Emil Krulish, P. A. Surgeon, U. S. Public Health Service, was written from Texas during the epidemic of cerebrospinal meningitis of 1912:—

The present epidemic of cerebrospinal meningitis in various portions of the State of Texas and the prevailing fear of its contagion by the public demonstrate that the question of prophylaxis is of most vital interest and importance. It is encouraging to those interested in public health work to note the interest that the public is taking in preventive medicine. The profession is consulted daily by the laity about some prophylactic which would ward off the disease.

All epidemics teach the inhabitants of the invaded sections that "prevention is better than cure," and the people of Texas have realized this fact, for wherever a case of meningitis has occurred the health authorities have improved the sanitary conditions of that locality. The late plague infection in San Francisco is responsible for the fact that it is considered today to be the cleanest city in the United States.

The preventive measures against cerebrospinal meningitis usually advocated by the profession are: isolation of patients and

contacts; prompt report of cases and suspects to the proper health authorities; disinfection of premises previously occupied by the diseased; closing up of places of public gatherings and antiseptic gargles and nasal sprays.

It is to the last measure, the application of antiseptics in any form to the nasopharynx as a preventive in cerebrospinal meningitis, that I take exception. I am of the opinion that these applications used promiscuously, as they are by the public, are worthless, if not directly injurious.

In order to direct a successful and an intelligent campaign against any epidemic it is essential for us to understand precisely the manner in which the disease is transmitted from one individual to another and, in infectious diseases, the method of invasion of the organism. Prior to the demonstration of Lazear and his associates that yellow fever was transmitted by the mosquito, our efforts to eradicate this disease were in vain. We know the exciting cause of cerebrospinal meningitis to be the *Diplococcus intracellularis meningitidis,* and it appears that the primary seat of attack is in the nasopharynx, but the connecting link between the nasopharynx and the spinal canal is still missing and until this is established we are at sea when considering the subject of prophylaxis. The meningococcus has been demonstrated in the secretions from the nasopharynx, both in typical cases of the disease, as well as in persons who developed no symptoms at all.

Advocates of the nasal spray presumably anticipate the destruction of the micro-organisms *in situ.* It is, however, obvious that any antiseptic in a solution sufficiently powerful to destroy these germs would be deleterious to the mucous membrane. The nasal chamber with its turbinate bodies and mucous membrane lined with ciliated epithelium is especially adapted to prevent germs from passing into the system, which is demonstrated by finding the tubercle bacillus, pneumococcus, diphtheria bacillus and, as previously stated, the meningococcus in the secretions of apparently healthy individuals. The nasal mucous membrane, therefore, possesses a high degree of phagocytic power.

The frequent spraying and douching of the nose has a tendency to injure this very delicate membrane and to destroy its function, thus defeating the primary object in view. The specialist has long recognized this fact and has discarded the use of the compression tank and pump.

It is an accepted fact that the exciting cause of phthisis is the tubercle bacillus, which is taken into the respiratory tract by inhalation through the nose, and one is almost constantly exposed to this infection, yet no one prescribes antiseptic nasal sprays for the prevention of tuberculosis; we endeavor to promote the function of the nasal mucous membrane and restore it to its normal condition if diseased.

Let us consider the use and results of the so-called antiseptic spray and douche. Spraying of the nose is accomplished by means of various forms of atomizers; their action is chemical or mechanical. The solutions recommended for this purpose are aqueous, as Dobell's, or oily, as liquid petrolatum, with eucalyptus or menthol. The oily solution is introduced into the nose in the form of a very fine vapor and, therefore, any benefit derived is necessarily chemical in nature, depending on its antiseptic properties. Taking for granted, however, that it is germicidal, we know from experience how difficult it is to reach the posterior portion of the nasal cavity with any form of a spray. The germs which may be present in these parts would, therefore, be beyond the firing line and remain undisturbed.

The aqueous solutions when used in the form of a spray can act but chemically, but if introduced in sufficient quantities or as a douche the chief action is mechanical; the germs may be dislodged and perhaps washed out with the fluid. But what actually occurs in the majority of cases is this: The bacteria are perhaps dislodged; the nasal cavity then contains more or less of the solution and the person invariably attempts to expel it by blowing the nose. Instead of closing one nostril and blowing the other, which is the proper way, the act is performed by closing the mouth and compressing both nostrils simultaneously, using the expanded lungs as a bellows. This forces the air up into the nasopharynx, where it is condensed under more or less pressure; the Eustachian tubes open, and some of the secretions with the organisms are very liable to be forced into the middle ear and perhaps into the accessory nasal sinuses. Thus the germs instead of being expelled are distributed through the head, where they usually find a favorable culture medium for their growth and infection is likely to develop.

If the theory of the nasal douche were efficacious, the postnasal douche would be the proper method of cleansing the nasal chamber and expelling organisms which may be present; a small catheter or

the tip of a syringe introduced through the mouth into the post-nasal space, and the solution allowed to flow through the cavity forward and out, thus actually washing *out* the nose. This method, of course, is not practical for the general use of the laity except under the direction of the physician.

Conclusions.

Nasal sprays as ordinarily used for prophylaxis against cerebrospinal meningitis are unnecessary because of the obscure etiology of the disease. They do not destroy the germs. It is impossible to reach every portion of the nasal chamber, and a normal nasopharynx is usually able to take care of itself. They are injurious from the fact that their use is likely to give the person a sense of false security and he is apt to consider other measures as being of minor importance; also the frequent use of the spray injures the delicate mucous membrane and lowers its vitality. Therefore, let us leave well enough alone as regards the nose. (Krulish, "Cerebrospinal Meningitis," Jour. Amer. Med. Assoc., Feb. 24, 1912.)

The physician who advocates and places much weight on nasal and buccal antisepsis as the only prophylactic measure called for during an epidemic of poliomyelitis should recall that, although such measures have been so generally accepted, the patent-drug man advertises a nose jelly as a sure preventive of infantile paralysis, yet this method of transmission of poliomyelitis by nasal and buccal secretions remains *unproven*. The flaw in the Flexner theory of nasal transmission is the failure to get secondary cases in institutions. I have many times observed that an influenzal infection once introduced into a hospital goes through it like a cyclone, invading the rooms of private patients with quite the same rapidity of progress as through the wards. There is also the known epidemiologic law that respiratory infections attain their maximum in the winter and closed-in season, while poliomyelitis has so far attained the maximum curve of incidence in the summer months.

PROPHYLAXIS FOR THE PHYSICIAN.

A very large number of cases have developed in the families of physicians attending acute poliomyelitis. Harbitz reports 9 cases which occurred in the household of one physician while attending cases. The following unusual suggestions may well have the consideration of the doctor attending these cases. The doctor's driving horses should have protection from flies. The doctor should request a plain wooden chair when in the sick chamber, and strike it on the floor before sitting down, ascertaining at the same time that his garments do not come in contact with the bed-hangings, from which bedbugs will crawl to the clothing of a fresh host with wonderful celerity and secrecy. The following routine prophylaxis was employed by Dr. Philip A. E. Sheppard, Special Investigator of Epidemic Poliomyelitis for the Massachusetts State Board of Health:—

(*a*) Washing of hands with soap and water before and after handling patients, excretions, etc.

(*b*) After handling patient and washing the hands, further rinsing in bichloride of mercury or equal parts of boric acid and chloride of lime.

(*c*) Final rinsing in alcohol, 70 per cent.

The following measures were carried out as often as practicable, especially before mixing in the company of healthy people:—

(*d*) Antiseptic mouth-wash and gargle with hydrogen peroxide or glycothymolin.

(*e*) Eye douche with boric acid.

(*f*) Nasal spray with menthol preparation.

Finally, after seeing the last case for the day, all clothes worn and other articles, including the bag in which they were carried, were placed in a sealed cupboard and exposed overnight to the influence of formaldehyde gas.

CHAPTER XI.

Treatment of Preparalytic Stage, and Progressive Ascending or Descending Paralysis with Impending Paralysis of Respiration.

TREATMENT OF PREPARALYTIC STAGE.

Treatment of the early stage of poliomyelitis is of grave importance and should not be delayed during the stage of invasion while the diagnosis is yet doubtful, for the diagnosis of this disease in many instances is not made, or made but tentatively, until the oncoming of paralysis. Much may be accomplished with a twelve-hour start of the paralysis, and should paresis already impend with one extremity presenting the aura of motor weakness, by effecting prompt and certain elimination and the dilution of the toxins distributed by the blood-stream to spinal fluid and ganglion cells, it may be possible to avert an ascending paralysis and death.

Recalling that a toxin may have instituted a destructive and constantly augmented action on the ganglion cells of the central nervous system, and realizing that every hour between the onset of poliomyelitis and the appearance of paralysis is a sixty-minute opportunity for preventing disability or death, the treatment to be promptly instituted, when symptoms may indicate and do not negative the stage of onset of poliomyelitis, consists of: isolation; confinement to bed; forced feeding of water; colonic flushings, repeated until elimination is established; application of ice to head and spine; evacuation of bladder; calomel; mag-

nesia; written orders, and *a check system to insure their faithful enforcement.*

In the majority of these cases seen first in the acute stage, while diagnosis may be doubtful, there is no question the patient is suffering from a profound toxemia. The child, the young male, as the case may be, is found with a flushed and strained countenance; head a trifle retracted: neck more or less rigid; muscles twitching; headache basilar, occipital, or very occasionally frontal, and a tender spine. There will be a history of change in disposition, tremor, inco-ordination, ataxia, a stumbling gait, or unaccustomed falls. There will be a complete paresis of digestion, evidenced by vomiting and obstipation, with abdominal pain and meteorism. The pulse rate will be much augmented and the temperature elevated, while respiration rate will be unaccountably increased. Some delay in voiding the urine always is present, retention is frequent, and retention with overflow may mask the paralysis of bladder.

What is first called for? Elimination. To secure intelligent co-operation from the parents or nurse this must be explained and forcibly impressed on them. Leave written orders, or, better, do not leave the case until elimination is established.

First Aid.—Forced feeding of water is first aid in these cases. Give an ounce or more of ice-water every fifteen minutes, remembering that at Hopkins they control temperature in the typhoid wards by the forced feeding of water. The forced feeding of water in the acute stage of poliomyelitis will

(*a*) dilute toxins in blood and spinal fluid which have been proven infectious during acute stage;

(*b*) decrease plus pressure of spinal fluid, by lowering specific gravity of blood and so checking hyperosmosis;

(*c*) wash out stomach by assisting the vomiting;
(*d*) flush kidneys, stimulate renal function, and increase amount of urine.

Further augment this flushing of the system with repeated colonic lavage, using pure water at body temperature with the addition of dissolved ivory soap if it becomes apparent that the digestive tube is paretic. The absorption of water at this stage is most important; toxins are promoting hyperosmosis from the nervous tissue; the spinal fluid has increased in quantity, until the pressure is unbearable. Novi maintained that the origin of uremic convulsions is due to withdrawal of water from the cortex cerebri. Reducing the specific gravity of the blood will at once set in action forces tending to check this osmosis. If no evacuation from the bowels is secured from these measures, a mixture of equal parts of glycerin and magnesium sulphate, added to the enema, will unload the lower bowel, and to cleanse the entire tract calomel should be used. An immediate effect is desired; therefore, give the full dose of calomel at once, 2 grains for a child, 3 to 5 grains for an adult; follow in one-half hour with the pediatrician's friend, carbonated effervescent citrate of magnesia; a feverish child will drink this greedily under the name of lemonade, and the castor-oil struggle has been avoided.

Examine the bladder; if there is distention, with inability to micturate, catheterize, unless other methods of relief have succeeded.

Place an ice-bag to the spine, and one well against the nape of the neck. Children with the burning fever of the onset of poliomyelitis find some relief from the ice applications, and will ask for the ice when it has been removed. Many of these children present the appearance of heat-stroke, and some of them have taken violent means to

secure relief from the fever in their veins, notably the 12-year-old boy reported by Dr. Marquardt, of La Crosse, who crawled under a garden sprinkler, and was found there in the acute stage of poliomyelitis. Pulmonary symptoms, beyond the increased respiration which is central, contraindicate any but the most intelligent use of the ice-bag in children.

An emergency ice-bag can be readily prepared from the inner tube of a worn-out automobile tire. Cut out a section, fill with ice, turn the ends over, and tie tightly.

GENERAL TREATMENT.

Isolation.—When poliomyelitis is suspected the patient should be promptly isolated and this isolation should be maintained in its entirety. The reasons for this isolation apply with equal force for the benefit of the individual himself, the family, and the community. It conduces to the quiet of the sickroom to permit no coming and going, and quiet is most desirable. Rigid exclusion will protect the patient from contact with any influenzal or respiratory infection, a dangerous and fatal complication with paralysis of the upper segment and chest muscles. While we remain in ignorance of the method of transmission of poliomyelitis, it is possible that the most innocent visitor should transmit the infection to a third person. Evidence accumulates that this disease is transmitted by an insect host, and, should that host prove to be the bedbug, any visitor to the sickroom might harbor and carry away the agent of transmission. It is believed by many physicians who have dealt with this disease in its epidemic form that isolation and postfumigation will check the spread of the disease.

The patient's room should be screened from flying insects, and bare and clean as a hospital ward; if climate

and season permit, the isolation is best carried out in a tent with a wood floor. A screened-in sleeping porch shares with a tent the great advantage of a constant supply of fresh air to lungs which may have lost more or less wholly their power of expansion from paresis of the respiratory muscles. The sudden advent of respiratory paralysis may occur in any case, and preparations for removal to fresh air should be adequate.

Rest.—Absolute rest in bed from onset of the disease is an important measure in the treatment of poliomyelitis. The mildest case may suffer a reinvasion of the attack, and the mortality rate is known to increase with exertion after onset. In many cases the prostration is so marked before appearance of paralysis that this measure is self-enforced. When otherwise, the infraction of the rule may be prevented by ascertaining that a bedpan, and not a commode, is provided, that drinking-water is conveniently placed on a chair or low table beside the patient, etc. The mere looking after such details by the physician impresses on the mind of the attendant that the orders are more than routine. When extreme restlessness is manifest, sedative measures of value are the employment of the ice-bag, cool sponging, immersion bath (given, not taken), and codeine.

Position.—A child will show gratitude for gentle changes in position, and it is questionable whether they should be allowed to rest continuously on the back, although many cases with the preceding pain in and paralysis of both legs prefer that position. A little girl, who objected to the slightest shifting to one side and support with a tucked-in pillow, allowed this change when an ice-bag was interposed between the spine and pillow, and a flat, soft pad was placed under the superimposed leg. Armstrong, of Minnesota, reports a boy of 5 years who for a week lay on

his face because it hurt his neck to lie on his back. The head may be arranged over a small, flat pad, in an extended position, and the warm pillow eliminated. Every case must be studied and its needs consulted in this matter of position, where groups of muscles are spastic from pain and exquisitely tender to pressure or touch. Do not make the mistake of applying any method of rigid immobilization to these tender and anguished frames. The inquisition furnished no more cruel measure than the application, which has been advocated, of a plaster jacket during the acute stage of poliomyelitis. It has been advocated to "immobilize the spine during the period of muscular excitability, as we would any acutely inflamed joint with muscular spasm." Some of these children lie rigid, "stiff as a log from head to heels," and the advantage of artificial immobilization is not obvious in these cases; the majority of children and adults present a beginning opisthotonos; the mildest form may be that of a nuchal rigidity, with the head slightly extended; the severe grade is hyperextension of spine to the bent-bow outline from head to buttocks, with extreme flexion of leg on thigh. If measures which assist relaxation are not promptly inaugurated, inexorable nature pursues her own method of relaxation and a flaccid paralysis ensues. Would "immobilization of the spine" secure such relaxation; or, indeed, would it lessen in any degree the convulsive twitching of the quadratus, or peroneal, or shoulder group of muscles?

The disadvantages of permitting a continuous dorsal decubitus are: a possible hypostatic congestion augmenting the spinal congestion of the lesion; the initiation of bed-sores, which have given serious trouble when trophic disturbance accompanied the motor and sensory changes, and the very real danger of hypostatic congestion of the lungs should respiratory paralysis present.

Air or water beds are desirable to minimize pain by distributing pressure, and thus secure the maximum of rest and sleep. If they cannot be obtained, it will be found of advantage to shift the position of the patient occasionally, giving support to the aching extremities while avoiding the use of heat-retaining feather pillows; to ascertain that only sufficient bed-covers are employed (and a sheet is sufficient on a warm day, while the temperature remains elevated); to suspend these coverings over a large cradle in case the weight is in the least irksome to the patient.

Exclusion of Light.—The majority of cases suffer from photophobia, which is easily understood when we recall the relative size of the optic nerve, its great peripheral terminations, and the fact that autopsies have shown that a majority of cases, even of the spinal type, present lesions of the cerebral cortex. Total blindness results in some of the encephalic cases. The exclusion of direct rays of light from the eyes of the patient should be accomplished by some other method than darkening the room, for the reason that a darkened room is usually accompanied with a stuffy atmosphere and little ventilation. It is well to place the bed with the headboard toward the windows which receive sunshine, and it is often sufficient and convenient to merely arrange the patient with head at the foot of the bed. Direct rays of light, whether natural or artificial, should not fall in the direction of the patient's vision. Movable screens and shades may be utilized in these arrangements. All draperies should be abolished from the sickroom.

Before going on to the medication of poliomyelitis, I would like to dwell a moment on the difficulty of obtaining the enforcement of such simple hygienic measures as are noted in the foregoing. Many good people consider that

the sick should be buried in bed-coverings, and particularly heap them on sick children to their torture in the effort to overcome the supposed deadly effect of a draught. I am reminded of the reply made to the frantic question of a scandalized spinster whose nurse had forcibly removed her bedgown and left her with a sheet as sole covering on a scorching August day. "Doctor," said the spinster, "won't I catch cold like this?" "Catch cold!" said the sweating physician, "Catch cold! My dear madam, put your feet on the mantelpiece and enjoy yourself!"

MEDICATION.

Elimination.—It has been shown that poliomyelitis attacks most readily those persons who are subject to constipation, and that the attack is limited to the arrested form of the disease in a large percentage who eliminate the toxin readily with a diarrheal discharge. All methods for unloading the accumulated waste of the system should be promptly employed at onset of poliomyelitis. It is possible that this eliminative process rids the system immediately of a considerable amount of the toxin whose lesions have been observed in the liver tissues and intestinal lymphatics. The evacuation of residue frees the system, permitting absorption of water and the prompt effect of medication.

Elimination may be freely induced by the use of gastric and colonic lavage, cathartics, and diuretics. The use of diaphoresis and diaphoretics is questionable, and is not advocated by the writer. We do not as yet know where we stand in regard to the vasomotor system in this disease. Sweating is a profound accompaniment of the onset in many cases, and is followed by extreme prostration. Not diaphoretics, but vasomotor constrictors, have been advanced as useful.

Colonic lavage should be prompt and thorough, and repeated daily throughout the fever stage.

Calomel, castor oil, and magnesia have all been employed in the initial catharsis. The daily use of citrated magnesia or uncombined cascara sagrada will tend to overcome the paresis of the bowels, which may be encountered for a considerable period of time.

Diuresis should be promoted during the fever stage and thereafter; the forced feeding of water alone may overcome the paretic retention usually observed. The bladder should be under careful observation. Some difficulty in voiding urine is present in even mild and arrested cases. Painful retention with overflow at the onset of this disease has been mistaken for intussusception and operated (Lovett). A few drops of sweet spirit of niter are often effective in the small child. The immersion bath is better. If instrumentation is called for, it is well to use a mild internal antiseptic combined with a mild diuretic and the forced feeding of water during the period in which catheterization is employed. No complicating cystitis has followed the usual aseptic instrumentation when the formula below was in use. It is a mild internal antiseptic and diuretic for use when the paretic retention of poliomyelitis demands instrumentation:—

℞ Fluidext. tritici repentis ..... ℥ij.
Acidi borici ..... ℥j.
Syrupi ..... ℥ss.
Aquæ menth. pip. .....q. s. ad ℥iv.
M. Sig.: One (1) teaspoonful in water every four hours for adults; children in proportion.

Strychnine.—There is perhaps no more frequent mistake made in the therapy of the acute stage of poliomyelitis than the use of physiologic doses of strychnine sulphate,

for the use of such doses is in this case the addition of a detonator to a high explosive, and highly destructive in its action.

The chief physiologic action of strychnine, according to the United States Dispensatory, is stimulation of the motor and vasomotor centers of the cord. It is cumulative in action, and an overdose results in tonic and clonic convulsions reflex in character of all voluntary muscles; the body is rigid, the limbs stiffly extended, and the chest locked in a general respiratory spasm; death occurs, with symptoms of asphyxia.

The above description is a fairly accurate picture of a severe case of poliomyelitis, and strychnine immediately augments the irritation which is already taking place in the ganglionic neuron. It has been a common thing to load a child up with hypodermics of strychnine every four hours during the acute stage of poliomyelitis. Frauenthal has seen 3 cases of children with strychnine poisoning from this treatment, and considers it probable that some deaths credited to infantile paralysis were directly caused by cardiac spasm induced by overdosage and the cumulative action of the strychnine prescribed. The exhibition of strychnine is invariably advocated by the consultant unacquainted with the lesions of this disease, while those authorities who have had the most extensive opportunity of observing cases during the acute stage of the disease are unanimous in condemning its use until the chronic stage is well established.

Bichloride of Mercury.—Hexamethylenamine has completely failed to arrest the inflammation in cord and meninges, although it has been thoroughly tested in the Washington epidemic. There is, however, a remedy of which we may hope that further trial will show the use. I refer to mercury, the power of which over some infec-

tions has become better realized since it has been employed by injection into the muscles or veins.

A Case.—I have, however, only had opportunity to test it in 1 case of poliomyelitis during the acute phase. It was done because of a sudden advance of acute ascending paralysis in an adult who had apparently improved on the preceding day, the fifth of his disease. By lumbar puncture 10 c.c. of cerebrospinal fluid were withdrawn. Headache and nausea were at once relieved, and the paralysis ceased to progress, while the temperature fell steadily. During three days, five doses were given of mercury bichloride of ⅓ and ¼ gr. alternately. In another case of the same kind with which I was associated, the remedy was not tried, and the disease progressed to respiratory paralysis and death on the fifth day. Both these cases were adults seen in consultation, the first with Dr. John Lewis, of Bethesda, Md.; the second with Drs. A. B. Hooe and Roy, of Washington. It is true that the injections would greatly perturb a child who is hyperexcitable from meningitis; but a temporary disturbance is preferable to paralysis or death, and much less pain is produced by the small needle used than by the injection-syringe needle required when diphtheria or meningococcus diseases are in question. If the disease is protozoal, the *rationale* of mercury is evident. (Williams, of Washington.)

Counterirritation.—Any method of counterirritation is contraindicated in poliomyelitis. Cauterization of the spine, and the application of mustard plasters, or fly-blisters, are methods especially pernicious during the acute stage, for at this period they augment the pain and render possible the formation of bed-sores. Death from sepsis followed the application of the medicated (?) spinal plaster used by a notorious faker of the West.

Vasomotor Control, Local Depletion, and Resorption. —Application of the d'Arsonval high-frequency current to the spine during the acute stage of poliomyelitis is used and recommended by Dr. Henry Frauenthal at the New York Hospital for Deformities. Observing that the primary effect of the application of the high-frequency current in obliterating endarteritis was a blanching of the skin which continued for several moments, the current was applied to the spine in the treatment of the early stage, with, in 1 case, inhibition of further advance of a progressive paralysis and rapid and complete regression. The d'Arsonval current in the hands of Arrhenius, of Stockholm, has recently been shown to exert an extraordinary stimulus to the metabolism of the living cell. The oscillating current may have a rate of alternation of millions a second. The molecular action on the body tissues, which are about nine-tenths water, is only surmised as yet. An agent which may produce local depletion cannot be overlooked until a specific for poliomyelitis has been discovered. The contraindication for the use of the high-frequency current is the augmentation of stimulation to the ganglion cells. A method of local depletion used with excellent effect in acute otitis media I have not seen advocated in poliomyelitis, but believe it well worth a trial. When Pasteur was attacked by the acute spinal paralysis which I have elsewhere shown to be an attack of poliomyelitis, his astonished and bewildered consultants applied sixteen leeches to the occipital and cervical region, believing they dealt with cerebral hemorrhage. In the treatment of an ascending paralysis, succeeding the establishment of thorough elimination, I should now endeavor to obtain local depletion and the determination of fresh blood to the cervical and upper dorsal region, by the application of several leeches.

Ergot and Gelsemium.—The use of ergot and gelsemium is advocated in poliomyelitis, to lessen the supply of blood to the congested area of the cord. Ergot, which was formerly said to have a selective action on congested pulmonary vessels and was used to abort pneumonia, is now called on to exert a similar selective action on the vascular supply of cord and brain. If such local action could be obtained it would be undesirable. It is not a lessening of the blood-supply that is needed, but more blood: fresh, pure, and regenerating. The study of a section from the lumbar level of the cord of an acute case of poliomyelitis is convincing in this respect. The cut ends of the vessels are not distended; they are choked with a collar of round cells; their walls have been rendered permeable; hyperosmosis is taking place; the walls are leaking at every joint. It is not desirable to cut off the supply of blood, but to augment it with a fresh and toxin-minus supply.

"Ergot, I believe, is contraindicated. I can see no reason why a drug which contracts the smaller arteries should be given in a condition in which the blood-supply of nervous centers is already diminished by a too small caliber of the smaller vessels." (Skoog.)

Antipyretics.—The temperature of poliomyelitis is a measure of the struggle of the organism against the toxin. It is best alleviated by hydrotherapy, external and internal. The forced feeding of water, colonic flushings, sponge baths, immersion baths when possible, and the ice-bag are all efficient aids. The temperature will not fall when the bowels are finally moved during the onset of the case, unless paralysis is impending, or the case is happily merging into the arrested type. Hydrotherapy will lower the temperature only temporarily until the fall by crisis which precedes the paralysis. Nevertheless, the measures relieve and benefit. The parents may request you to break the fever; they may have been told by an unscrupulous

confrère that the fever should be broken. Explain to them that any drug sufficiently depressant to the heart's action to lower the temperature of the child at this stage will weaken the heart and lessen the chances for life and a fortunate issue.

Sedatives and Analgesics.—The foregoing remarks about antipyretic drugs during the acute stage of poliomyelitis apply equally well to the administration of salicylates: they are equally depressing in effect, and their exhibition in these cases I consider dangerous. Bromides, in addition to their depressant action, are irritant to the stomach, which should be humored into retaining as much nourishment as possible. With extreme restlessness, or pain, or both, codeine is the most soothing and least harmful drug which we have for these cases. Morphine, or other preparations of opium, may be used with great caution as rectal suppositories, remembering that their use may undo all you have striven to attain, by again checking elimination.

Internal Antiseptics.—The internal administration of some preparation of formalin has been advocated during the acute stage of poliomyelitis, and its usefulness would seem to be demonstrated by the fact that the presence of hexamethylenamine has been demonstrated in the spinal fluid of monkeys one-half hour after administration. The liberated drug is said to have an inhibitory effect on the toxin in the cord, and we must therefore examine its advantages and disadvantages, to make use of or have excellent reasons for discarding such a powerful medicament.

Before summarizing the advantages and disadvantages attending the use of the many mercantile preparations of this drug, we may consider the statements of physicians making use of this drug:—

Hexamethylenamine (urotropin) may have some effect in disinfecting the spinal fluid, but if long continued there is a possibility that the formalin set free may have a hardening effect upon the spinal cord. Two grains may be given every two hours during the first two or three days. (Wm. Spiller, Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania, "Diagnosis and Medical Treatment of Poliomyelitis," Pennsylvania Medical Journal, Dec., 1911.)

Urotropin was sometimes given and with uncertain results. (Anderson, Nebraska, "Epidemic of 279 Cases of Poliomyelitis," Pediatrics, August, 1910.)

Urotropin should be given in full doses well diluted and kept up for quite a while, but watch out for irritation in the urinary tract. (Marvin, Discussion of paper, "Infantile Paralysis," by Thompson, Kentucky, Louisville Monthly Journal, April, 1912.)

It therefore seems a logical remedy (hexamethylenamine) in acute poliomyelitis, the dose to be from 5 to 15 grains every three hours according to the age of the patient, the medication to be instituted at the earliest possible moment. (Bierring, "Acute Poliomyelitis in Iowa," Interstate Medical Journal, Jan., 1912.)

While there is no proof as yet that this drug (urotropin) has any effect in modifying the course of the disease, its use is free from any valid objection and is quite generally recommended. (Frost, United States Public Health Service Bulletin, No. 44.)

Fullerton reports a case of medicinal cystitis following its use, with painful, burning, frequent urination, hematuria, and the passing of many small blood-clots as well as pieces of bladder-membrane several centimeters square. For two nights and three days after the onset of the hematuria the patient continued to pass blood-clots and bladder-membrane. The urine was demonstrated to be sterile; the symptoms arose after the drug was begun, and cleared up at once, though gradually, after it was discontinued, and, there being no other possible etiologic factor, all this seems to be conclusive evidence that this was a medicinal cystitis.

It has also been reported that the injection of hexamethylenamine in guinea-pigs produced congestion and hemorrhages into the stomach.

Is the mucous membrane of a child's stomach more resistant than that of a guinea-pig? Is the delicate vascu-

lar lining of a child's bladder less susceptible than that of a woman of 25 years of age? Are we to empirically liberate in the spinal canal a solution known to have a hardening effect on living tissue-cells? In poliomyelitis, with a destructive toxin already at work, are we justified in introducing a killing and fixing agent into the spinal canal? If the drug is not liberated, is the medication of value? These are questions every practitioner should consider before making use of the various preparations of formalin that are at present advocated for poliomyelitis in children.

The use of hexamethylenetetramine in poliomyelitis was first advocated, if I am not mistaken, by Preble, of Chicago, "in view of our desperate helplessness," in a letter to the Journal of the American Medical Association. Those who advocate its use today claim hexamethylenamine (*a*) will break down and liberate free formalin in the tissues. (*b*) If given by mouth will remain in blood, bile, and gall-bladder for twenty-four hours. (*c*) If the dose is so much as 75 grains per day, it will prevent bacterial growth in these passages (Crow). (*d*) It may remain unchanged on excretion, only liberating formaldehyde after more or less stagnation. (*e*) It is excreted in the urine unchanged, and breaks down after remaining in the bladder at least one and a half hours. (*f*) It is recommended in genito-urinary affections when catheterization has to be resorted to for any length of time. (*g*) Free formalin has been demonstrated in the spinal fluid of apes one-half hour after administration.

Dosage and Administration.—Spiller prescribes 2 grains of urotropin every two hours for two or three days only. I have seen no report which justifies the use of a larger dosage for children. The drug is administered after solution in a large amount of water, and is said by

Ager, of Brooklyn, to be less irritant when accompanied by Vichy or bicarbonate of soda.

Quinine.—Hughes, of St. Louis, advocates a vigorous course of quinine in poliomyelitis. Quinine sulphate is a powerful internal antiseptic and well tolerated by the economy. The blood during the acute stage of poliomyelitis is known to be infective. Lacking a specific treatment in poliomyelitis, the use of quinine, while empiric, is justifiable.

Echinacea.—The exhibition of echinacea during the acute stage of poliomyelitis is advocated by Frauenthal, who considers it to be the best of internal and local antiseptic. The dosage is 5 minims to 1 dram of the tincture of echinacea every six hours. Locally it may be applied in full strength or diluted in 1, 2 or 3 parts of water.

Lumbar Puncture During Acute Stage of Poliomyelitis.—There are three methods by which pressure may be reduced and toxicity lessened during the acute stage of poliomyelitis:—

1. Elimination (previously outlined).

2. Dilution and lowering of specific gravity of blood (previously outlined).

3. Lumbar puncture.

A quantitative increase in the cerebrospinal fluid is constant during the acute onset of poliomyelitis, and is evidenced by a plus pressure or the actual spurting of the spinal fluid on lumbar puncture, as well as bulging of the fontanels, and Macewen's sign.

The cerebrospinal fluid drawn during the acute stage has been proven infectious. Theoretically it would seem the abstraction of an amount of this fluid would relieve tension and remove toxic material. The majority of recent reports on lumbar puncture during the acute stage of poliomyelitis, whether the tapping was done for diagnosis or

treatment, indicate that the method is of therapeutic value. Lumbar puncture is also a valuable and only certain method of diagnosis of the disease during onset, and of prognosis as to the progression and regression of the paralysis.

Routine Method of Lumbar Puncture of New York Health Department.—Patient in left lateral position with flexed spine. Paint cutaneous area over lumbar spines with tincture of iodine. Freeze surface with ethyl chloride spray. Puncture in *median line* between fourth and fifth lumbar spine.

The possibility of a needle breaking during lumbar puncture may be averted by the use of a simple apparatus devised by Lorenz, of Wisconsin. A light padded stick placed beneath the knees, and attached by means of light straps to canvas bands crossing the shoulder and beneath the armpit, and drawn close, will secure the flexion desired and prevent sudden extension.

Objections to the use of lumbar puncture are the possibility of (*a*) sudden death; (*b*) infection; (*c*) the breaking of a needle, previously noted; (*d*) friends of patient attributing paralysis to puncture; (*e*) hemorrhage from puncture of a vessel of the pia-arachnoid.

Hansen, of Christiania, has recently collected reports of 30 deaths following lumbar puncture. As lumbar puncture has been in quite general use since the introduction of spinal anesthesia in 1899, the total percentage of fatalities must be very low.

The danger from infection might increase with repeated punctures with a lessening of surgical technique.

The patient's friends should be notified, before the procedure, that paralysis is looked for, which the puncture may avert; they will otherwise, if ignorant, almost certainly attribute the paralysis to the treatment.

Shidler, of Nebraska, reports that in 2 cases of the

neuritic type unrelieved by large doses of opiates the pain was lessened by lumbar puncture.

Spiller advocates lumbar puncture for the relief of the intractable pain, stating that he has known lumbar puncture to give great relief under such circumstances.

Serologic Treatment.—The serum treatment of poliomyelitis is still in the experimental stage. Drs. Anderson and Frost demonstrated a serologic neutralization test of much value in the determination of cases of the arrested (abortive) type. Serologic immunization of monkeys offers hope that human immunity may yet be artificially obtained. There is at present no antitoxin for poliomyelitis.

Diet.—The diet should be restricted, nutritious, and easily digestible. All fruits should be cooked, and no seeded berries, etc., given, to clog and irritate a paretic digestive tube. During the fever stage a child will take ices and iced drinks freely. Fruit juices and ices should be left pleasantly acid. Lemonade, orangeade, pineappleade, and the same ices are acceptable.

If the patient is not fond of milk, it may be rendered very attractive when served as one of many cream soups or broths. Corn soup, which consists of milk heated with canned corn, or freshly popped popcorn strained and seasoned, is a dish that many children who dislike milk will take eagerly. Croutons or educator crackers add to the nutritive values. Fresh chicken broth, with or without the addition of rich milk, served with soda crackers or toast, is much relished. Milk toast, a dish consisting of a plate of fresh and fragrant toast, and a pitcher of scalding, creamy milk, to be poured over the toast slice by slice at the bedside, where the child may inhale and be tempted by the appetizing aroma of both, is a much more nutritious dish than the sodden mass of charred bread and white

sauce frequently presented the sick. Oyster soup, clam broth, and, where it can be obtained, coquina broth are all appetizing and nutritive dishes.

All of the cooked breakfast foods may be used as such, or, slightly sweetened, molded and served cold with cream. Rice is of value. The package rice is a larger, cleaner kernel than ordinary bulk rice. It should be washed through numerous waters, and cooked in boiling water until tender, then drained and steamed till each kernel is a separate entity. Served with a separate dish of steamed dates and rich milk, the food values are in perfect combination.

Many of these children will have to be fed. When the arms are affected it is usually the right arm that is paralyzed. When the legs only are affected the child from pain or prostration may leave the food uneaten. The feeding should be unhurried. When there is difficulty in drinking, it may take considerable ingenuity to bend a glass tube at just the right angle to reach from the child's mouth to a glass arranged just below the pillow, but it can be done, and the reward comes in seeing the child gradually absorb glass after glass of the water, etc., with no extra exertion.

In paralysis of the pharyngeal muscles it may be necessary to resort to stomach feeding. Regurgitation of liquids through the nose indicates some beginning paresis.

TREATMENT OF PROGRESSIVE ASCENDING OR DESCENDING PARALYSIS WITH IMPENDING PARALYSIS OF RESPIRATION.

I. Remove patient to outdoor air. Porch; tent; roof.

II. Use pulmotor.

III. Use Sylvester method of artificial respiration.

(NOTE.—A satisfactory pulmotor has been devised from a vacuum cleaner. Langhorst, Elmhurst, Illinois.)

CHAPTER XII.

Hydrotherapy in Infantile Paralysis.

THE *value of hydrotherapy in poliomyelitis,* in conjunction with other forms of treatment herein described, compels serious consideration. We regard such measures as a part of the remedial armamentarium from the onset of the disease.

Fever is one constant symptom of onset of the acute attack. It varies from an elevation of 100° to 103° F., and the initial rise probably averages 104° to 105° F.

Cold sponging, augmented by the application of a cold pack to the spinal column, may be used to allay the moderate fever. With a more marked elevation of temperature and especially with those cases occurring during the extreme heated period of summer, the immersion bath should be used. That many such cases instinctively seek the immersion bath is evidenced by the number of histories of cases who have been in swimming a few hours preceding the appearance of the paralysis. When the preparalytic temperature remains continuously at a considerable elevation, 105° or above, it is well to employ the continuous suspension bath until such time as the fever gives indication of terminating by crisis. This continuous bath was also instinctively sought by the 12-year-old patient of Dr. Marquardt's, who crawled under the garden hose while in the acute stage and was found there some hours later.

The cool immersion bath aids in controlling temperature by the abstraction and diffusion of heat, and thus assists and renews the body tissues in their warfare against the destructive virus.

The bath also relieves pain in some degree and may banish it temporarily by providing a buoyant supporting

medium which equalizes and distributes pressure, this relief conducing to at least brief relaxation and rest.

Other hydrotherapeutic measures of great value during the onset of poliomyelitis relate to the forced feeding of water, the use of repeated colonic lavage, and the application of ice to the spinal column; these measures are given in detail in the chapter on the Treatment of the Preparalytic Stage.

The agonizing hyperesthesias present necessitate the intelligent, steady, and firm handling of these patients during the administration of pack or bath. The most painful area is the spastic neck and spine, while the muscles of the buttocks and hip of the leg which will later become paretic are frequently as tender as the area about an abscess. Avoid flexing the head on the chest, the leg on the thigh, or the thigh on the abdomen; use most care in not antagonizing the rigid spine to the great agony of the patient by attempting to lift or carry in the usual manner with one arm under the shoulders and one under the knees.

A portable bathtub, drawn to the bedside, and a bed lift with pulley and tackle, with a rigid framework on which the patient reclines, supply the best means of transportation between bed and bath. The young patient should not be left unattended in the bath for a single moment of time, and as several other matters must be arranged during this brief period, such as maintaining the temperature of the water at a uniform heat, it is desirable to have an assistant during the bath period.

With the approach of the paralysis the fever declines by crisis and the body temperature frequently becomes subnormal, while the paralyzed extremity becomes flaccid, cold, and clammy.

At this period the immersion bath should be given at

a temperature of 100° to 103° F., beginning each bath at the former temperature and gradually raising the water's warmth several degrees. The pain, which is still present in this stage, is very amenable to heat applied by means of hot-water bags, the electric pad, the hot sand-bag, etc.; but the most immediate relief with subsequent rest is found in the use of the hot immersion bath. Fifteen minutes are sufficient to allow for the bath, after which the child should be lifted out on a dry blanket and the drying of body done in bed. The temperature of the room should prohibit any postlavage chilling.

The most important function of the bath during this and the succeeding chronic stage of infantile paralysis is no doubt overlooked. I wish to call attention to it with some emphasis. When the paralyzed limb is lowered into the water for some minutes the surface of the limb becomes accustomed to the water pressure, which is sixty-two and a half pounds to the square inch; after removal from the water into the atmospheric pressure, which is fifteen pounds to the square inch, the change in pressure results in a type of hyperemia that continues to act for some time. The nutritional value of this hyperemia when regularly induced will tend to counteract the trophic loss of the lesion. I consider it to be of great value in conserving the nutrition not only of the superficial muscular tissue, but with a possible tonus for the proliferating osseous tissues.

As soon as pain has ceased to be a constant factor in the paralyzed extremities, the attendant may begin passive movements. These movements are most easily undertaken while the child is immersed in water of a temperature to slightly relax and sooth all hyperesthesias, and when the child's confidence has been established they may be continued when out of the bath. Valuable when the limb cannot be manipulated by the little patient, they are yet not to

be compared in value with active movement spontaneously produced by the child himself.

With the increased ease of movement which comes from suspension in water, the child should be induced to make free active movements; this is perhaps most easily effected with games connected with the delightful expanding and floating toys devised by the Japanese. An aquarium one day, a floating garden the next, and some ingenuity combined with much intelligence on the part of the attendant will be rewarded with the utilization of all uninvolved musculature. Amusement should never be allowed to grow trite, nor the bath continued above one-half hour. Kicking and splashing should be encouraged, with constant supervision that disabled cervical muscles do not allow sagging of the head to the water line. Tact must terminate the bath in a happy manner that will avert disturbance.

WARM-WATER BATH FOR PARALYTIC LIMB AT BEDTIME.

The circulation of the paralytic extremity is always impaired and the child suffers from cold. The limb will be found to have a surface temperature several degrees below that of its fellow. A warm-water bath at bedtime, during which the limb is immersed in water between 100° and 110° for twenty minutes, will stimulate the circulation and the member will be found to remain warm for a number of hours thereafter. This treatment, while adding greatly to the comfort of the patient, brings about an actual trophic change which stimulates growth in the tissues and bony structures.

The addition of sea-salt to the water of the bath will find favor with parents, and as the addition renders the water slightly more buoyant there may be some slight

value in its use. The daily bath should be followed by a nap, and on sunny days by a sun bath. The sun bath should be given by direct exposure of the body to the sun's rays; with summer heat this can be easily attained; later in the season a sunny window and heating plant are necessary paraphernalia of the sun bath. Protect the eyes if there is any complicating photophobia.

Guard against chilling after the bath. The paralyzed limb may be wrapped in soft flannel; an electric pad will be found a desirable permanent occupant of the side of the bed, and will prevent continual refilling of hot-water bottles, with possibility of a spill.

The child should never be left alone for one instant in the bath, for fear of drowning. The sun bath may be replaced by dry heat, baking the extremity in hot air, etc.

CHAPTER XIII.

Electrotherapy.

THE therapeutic use of electricity has already as wide a range as the therapy of water, and the degree of utilization of these great agents varies directly with the period of time which has elapsed since the date of discovery of their usefulness. The human race for untold centuries has been acquainting itself with the therapeutic values of visible and palpable water. The far more powerful agent, electricity, unrecognized because unseen and unfelt, has only recently been harnessed to the uses of humanity, who now begin to perceive the energy, the initiative to cell growth, the inhibition of destructive tissue change, the vital heat, and the remedial light to be derived from it. We know that 90 per cent. of the body tissues are composed of water, but are unaware of the proportionate values of energy, initiative, temperature, and cell growth, and their possible maintenance by somatic electric manufacture, storage, and discharge.

The methods of electric treatment of the acute and chronic stages of poliomyelitis differ absolutely as to the conditions encountered, the results to be achieved, and the form of electricity to be employed. The physician may call to his aid the high-frequency current, the static machine, the X-ray, the galvanic and faradic currents separate or in combination, or the concentrated sunshine of the leucodescent lamp. By these various agents he may successfully relieve pain, produce resorption, stimulate vasomotor con-

traction, maintain muscular tonus, promote cell metabolism, and stimulate defective growth.

Electric energy is regarded as life, and considered to

Fig. 71.—Total regressive paralysis of all four extremities, with atrophy and normal function. No early treatment. (N. Y. Hospital for Deformities and Joint Diseases.)

be the hidden force that actuates cell vitality, by many of those investigators whose research best qualify them to speak of its manifold modes of power and expression.

While the blood-vessels of the spinal cord are the pri-

mary point of attack of the toxic virus of poliomyelitis, the inflammatory processes rapidly involve the meninges, the gray motor and the white conduction tracts, all nerve-elements being successively reduced by pressure or starvation to a state of extreme irritability, *suspended animation,* or necrosis with the formation of scar tissue.

Fig. 72.—Total regressive paralysis of all four extremities with treatment initiated during acute stage. Perfect function, no atrophy. (N. Y. Hospital for Deformities and Joint Diseases.)

While motor paralysis is the more dramatic effect of the lesions, the augmentation of the pain sense, the impairment of tactile and temperature sense, the implication of the co-ordinating centers, and the retardation in developmental processes, when overlooked or ignored, are responsible for many failures in treatment.

In consideration of the motor paralysis it was well said:—

Treatment directed solely to mechanical ends—whether by correcting deformities or strengthening individual muscles—cannot of itself suffice for the requirements of all cases.

1. The (palsied) limb may be considerably smaller than its fellow without much impairment of its motor function.

2. With severe palsy the limb may nevertheless remain of normal length, keeping pace with the sound side.

3. In the case of a diseased upper segment—say, a thigh—the lower segment (leg) may grow longer than the corresponding portion of the undamaged limb.

In electricity we have the *only agent capable of stimulating defective growth;* this fact alone places those who use it with skill in a position of peculiar strength. (Hernaman-Johnson, British Jour. Children's Dis., December, 1911.)

HIGH-FREQUENCY CURRENT DURING ACUTE STAGE.

Application of the high-frequency current to the cutaneous surfaces of the spinal column is recommended at the earliest moment after motor inco-ordination, or other signs or symptoms, confirm a suspected diagnosis of the onset of infantile paralysis. The current is to be applied to the point of toleration, for five minutes, twice daily. This treatment stimulates vasomotor contraction of the hyperemic vessels, tends to reduce or prevent further extravasation of their fluid contents, sterilizes the infected tissues, and inhibits the progress of the ascending lesion.

I have found that, in the application of the Oudin or d'Arsonval high-frequency current in cases of obliterating endarteritis, the skin became blanched and remained so for several minutes, showing that the primary effect of the high frequency is a contraction of the blood-vessels. With this effect in view, hoping to reduce the blood and serum compression on the nerve-cells in the spinal cord, I have

in the first few days of the paralysis applied this current along the spinal column, irrespective of temperature or other symptoms, and feel positive that I have relieved the compression in the cord and hastened recovery. I will cite one case of many:—

Fig. 73.—Infantile paralysis. Gertrude G.

Gertrude G., 6 years of age; normal delivery; previous history not pertinent. I was called by Dr. Brainglass to confirm his diagnosis of infantile paralysis, on June 13, 1910. Sister of the patient had had the disease with facial involvement.

When I saw this child both legs were involved; the paralysis was extending, and seemed to be of the progressive type. Hoping

to stay the advance of the paralysis and expedite a recovery, having explained to the doctor the benefit I hoped to obtain by the d'Arsonval current, and receiving the concurrence of the family, I had the child removed to the Hospital for Deformities and Joint Diseases, where, after the first two days' treatment with high frequency along the spine, particularly over the lumbar region, a marked improvement in the child's condition with no advance of the paralysis was shown. This treatment was kept up and combined with the other treatment of massage and electricity.

The child has made an almost perfect recovery; can bear her weight and hop on either leg, and walks with a normal gait.

The apparent arrest in progression of an ascending paralysis obtained by the local application of the high-frequency current in this case is the result also sought in recent experiments as to the effect of subdural injections of epinephrin in the acute stage of poliomyelitis.

In the treatment of cases during the summer of 1907 and 1908, I secured results from the application of the high-frequency current that I failed to secure from the use of adrenalin or ergot, and so reported to the American Electrotherapeutical Association at the annual meeting, September 19, 1908. (Frauenthal, "Anterior Poliomyelitis," Jour. of Adv. Therapeutics, May, 1909.)

I cannot attribute the results obtained wholly to the vasoconstrictor action of the high-frequency current, and consider that the vessel walls are rendered less permeable, and that the transudation of fluids and the migration of cells are checked, by a direct oxygenating and vivifying action of the venous blood. I consider this oxygenation of sufficient power and extent to partly or wholly sterilize the invaded tissues in a similar manner to the electric sterilization of water.

The sterilization of drinking-water is secured by constantly passing the water between highly charged metal plates, whose electrification liberates oxygen so rapidly

that an ozonation of the water incompatible with bacterial existence results. This system of sterilization of water is so far superior to any other known method that it is in use

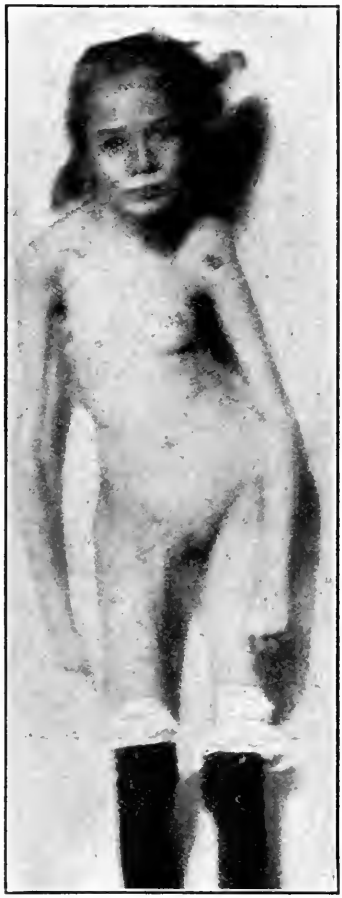


Fig. 74.—Infantile paralysis. (N. Y. Hospital for Deformities and Joint Diseases.)

Fig. 75.—Same as Fig. 74, posterior view.

in many municipal water plants on the Continent, and has recently been installed by the United States Government in the Philippines, where it has been demonstrated to decrease bacteria and the virulence of toxins.

Case referred to the Hospital for Deformities and Joint Diseases by Dr. Reginald H. Sayre, who took the original photographs. This case suffered from a severe attack of infantile paralysis in the epidemic of 1907. The *original in diagnosis*, from pain, was *rheumatism*, and *this diagnosis was concurred by*

Fig. 76.—Same as Fig. 74, one year later.

Fig. 77.—Same as Fig. 76, posterior view.

consultants. Her total paralysis was not noticed until two weeks after the original attack. After six months she was referred to an orthopedic specialist, and she journeyed from Yonkers to New York once a week to receive treatment, which consisted of electricity. The journey back and forth completely exhausted her for twenty-four hours.

The curvature in her back became so pronounced that a brace was applied. The deformity progressing, another consultation, with

a second orthopedic surgeon, was requested. As a result of this consultation a Calot jacket was applied; was also wearing a brace on her right foot. No improvement was noticed.

Consultation with a third orthopedist was sought. This orthopedist endorsed the treatment as conducted.

In September, 1909, she was seen in consultation by Dr. R. H. Sayre, who recorded her condition in Figs. 74 and 75. Regarding her case as one that should receive aggressive treatment, she was turned over to me, at the hospital, where for three months traction was made on her extremities and head, with daily electricity and massage to her weakened muscles. This was followed by a plaster jacket and jury-mast.

Photographs taken one year later, as shown in Figs. 76 and 77, show marked improvement; also demonstrate the fact, not accepted by many orthopedists, that in rotary lateral curvature, with bony deformity, much improvement can take place. A line drawn down from the left nipple in the front, or from the angle of the scapula in the back, or following the crease of buttocks up, will bring out the change that has taken place.

Since these photographs were taken, the patient has made decided improvement, but objects to having another photograph taken.

THE COMBINED GALVANIC AND FARADIC CURRENT IN THE EARLY TREATMENT OF PARALYSIS.

The usual advice of those practitioners who advocate the use of electricity in palsied limbs is to postpone treatment for a period of six or eight weeks after the subsidence of the acute stage, or at least wait for defervescence. They argue that the stimulation of the exhausted neuron by any agent, strychnine or electricity, is as ill-advised as the flogging of a dying horse.

The first requisite of electrotherapy in paralysis is the maintenance of muscular integrity during the period in which the injured neurons remain in a state of suspended animation, or until contiguous nerve-fibers take on the function of necrosed ganglionic units. Advanced muscular

atrophy is a not uncommon discovery at the close of the second week after the advent of paralysis. Is it good therapy to allow a degeneration to proceed for six weeks which will hardly permit of regeneration in a year's time, if at all? The treatment at this period should seek to provide rest, recuperation, and nutrition for the injured neurons; should minimize pain, correct postural deformities, and maintain muscular integrity.

It is during this period, while hyperesthesias interdict massage and passive motion, that a mild current, applied only to the origin and insertion of the affected muscles, for brief periods, once daily, offers the only known means of inhibiting muscular atrophy until such time as a beginning regeneration of the nerve-supply of the part obtains and peripheral function comes under the control of the will.

The rule of the Board of Health of New York City, which makes compulsory the isolation for six weeks of the acute case, should not be allowed to interfere with treatment at this important period.

We know that we can obtain contraction of the muscle by means of electric current when none can be obtained by the will; hence, this is a valuable means of retaining and developing the muscle fiber until it comes under the guidance of the mind.

When the temperature is between 98.5° and 99.5° and in some cases where high temperature continues for several weeks, I have not let this deter me from treating the patient.

I give to the muscles involved a sinusoidal current, alternating every second day with a combined galvanic and faradic current that contracts seventy-two times to a minute, synchronous with the heart beat (such a clock arrangement is found on Victor electric plates, and others); and I personally regard this as an aid in the effect.

Although many differences of opinion prevail as to the application of the sponge electrodes, I am in the habit of applying them at the origin and insertion of the muscle or muscle groups involved, always laying stress on the importance of approximating the origin and insertion of the muscle as nearly as possible.

For instance, in treating the perineal group, these muscles being most frequently involved, one sponge is

Fig. 78.—No treatment for fourteen years with flail arm and total disability. Result after nine months' electrotherapy, massage, and muscle training, a restoration of function of hand, forearm, and arm. (N. Y. Hospital for Deformities and Joint Diseases.)

placed over the middle third of the outer side of the fibula, the foot flexed as much above a right angle as possible, and the other sponge applied over the insertion of these muscles on the outer side of the foot. In this way the bellies of the muscles are relaxed and a contraction is made more easily for the patient.

The strength of the current used should be the weakest that will produce contraction, and it is never to be used

after contraction of the muscle ceases, nor longer than from two to three minutes on any particular muscle group, or from six to ten minutes on the body at one *séance*.

If this method is followed the child will not cry from pain, nor have its nervous system upset by too long continued electric treatment.

The blanket therapy of applying a large electrode to the spine and passing the second electrode over the entire surface of the involved limb, we consider harmful, at least during this stage of the lesion; it is usually painful, and greatly augments the pain of the hyperesthetic case.

The sponge electrodes are to be kept constantly damp, and a square of gauze interposed between the electrode and the cutaneous surface; this latter precaution is to be scrupulously observed when successive cases are under treatment.

Tactfulness is a large asset in giving electric treatment to young children; if the child's confidence is gained at once, it will not object to subsequent handling. The current should be turned off until the electrodes are placed and then turned on with entire gentleness only until a contraction is perceptible to palpation, by the index finger placed over the belly of the muscle. Avoid raising and replacing electrodes, as each break is painful to the hyperesthetic case. Time the treatment with a clock and do not exceed six minutes with children, nor twelve with adults, at this early stage. These periods of time cover the whole treatment with a proportional amount of time for each extremity, *i.e.*, three minutes' application to an arm or a leg is ample. A more extended treatment may be harmful. A deleterious result would follow the treatment recently advocated by a *confrère:* "Successively lengthen period (of electric treatment) from twenty-five minutes to an hour's duration" (!).

The mild treatment just outlined will frequently prove restful to the worn and helpless sufferer, and will induce the first restful sleep he has experienced since becoming ill.

If medication by strychnine is desired, the drug can be used locally and driven directly into the muscle and nerve-

Fig. 79.—Perfect result following treatment at N. Y. Hospital for Deformities and Joint Diseases.

endings during the electric treatment. Wet the electrodes in a solution of strychnine, $\frac{1}{10}$ grain to 1 ounce of water. A more simple and less dangerous method than the hypodermic injection of strychnine in young children.

"The muscles that are not affected are frequently treated, by the person using the electricity, to the detriment of the patient by

increasing the contractures. The paralyzed muscles will not respond to the electric current; the current is increased, and by extrapolar diffusion of the current muscles not paralyzed are made to contract again and again and contracture and deformity may be increased. The physician not thinking of the physiologic anatomy treats the muscles unaffected more than those which are really affected." (Mills, Philadelphia.)

M. M. Acute attack August 16, 1910. Paralysis of both lower extremities, back, right upper lid, and crossed eyes. First physician called did not know what was the matter with her according to the mother, who took the child to second physician (Koplik), who applied cups three times a week for six weeks. In November the child was removed to Hospital for Deformities. Under treatment there was steady renewal of function, and in January, 1911, the child began to walk; a perfect result followed.

ELECTRIC TREATMENT OF THE CHRONIC PARALYTIC AND ATROPHIC STAGE.

"By a proper appreciation of the available therapeutic and mechanical agencies we need rarely if ever encounter any paralytic deformity." ("Paralytic Treatment," page 41, Jones, Liverpool.)

This has been our experience with cases placed early under our care. Many contractions and deformities are preventable, first, by the maintenance of the integrity of muscle structure where weakness would allow overaction on the part of unaffected opponents, and, second, by the use of plastic splints. Every muscle not hopelessly fibrosed should be given a course of electric stimulation for an indefinite period of time. If a case of recent or long standing present with a paresis or atrophy of however severe degree, if extreme deformity or disability demand orthopedic intervention, or if continuous sensory disturbances combined with motor weakness on exertion are the only reminders of the acute attack, electrification of nerve and muscle is the valuable adjuvant to other treatment.

Electric stimulation of the ganglionic neuron through its peripheral branch directly improves local nutrition, in-

creases the caliber of the nerve of conduction, manifolds energy transmission, thus securing compensation for the decrease in muscle mass.

Fig. 80.—Acute ascending paralysis, with paralysis of diaphragm. (N. Y. Hospital for Deformities and Joint Diseases.)

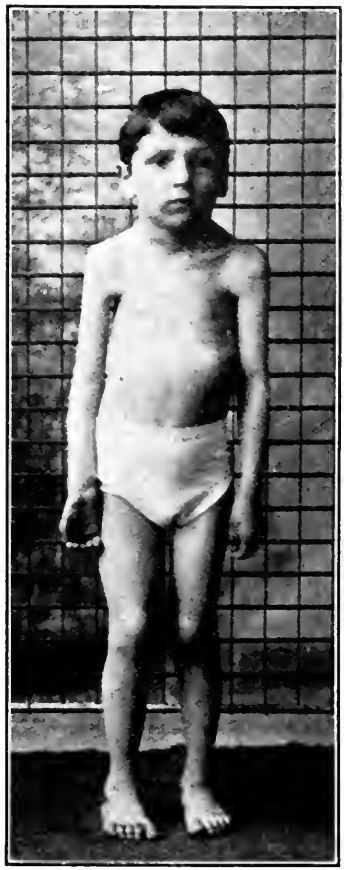


Fig. 81.—Same case as Fig. 80. Recovery.

The regeneration of nerves is apparent in the class of cases which present extreme muscular atrophy, yet regain excellent function under treatment; this regeneration may

take the form of an increase in calibration of the nerve, implying an increase in power of transmission similar to the increased transmission of an amplified copper wire; the regeneration may be due to the taking over of the function of one impaired neuron by a healthy neuron at a lower or higher uninvaded level of the cord. This is based on the fact that in investigation of localization in the brain and the decussation of the fibers, lower areas have been found to take over function of higher levels impaired by injury or tumor growth.

Mannie. Acute ascending paralysis, with paralysis of the diaphragm, and recovery. This case was referred to the hospital by Dr. Koplik seven months subsequent to initial paralysis. When admitted there was paralysis and atrophy of all extremities, with involvement of the pectorals, intercostals, and diaphragm. The right arm and hand presented spasticity, while balance of muscles were flaccid. The stage was considered a terminal one and an autopsy expected. Reflexes exaggerated on right side; lost on left side. Under treatment, there was a slow return to function. At the close of three years' continuous treatment with massage, electricity, breathing exercises, and muscle reduction the muscles of both legs have regained normal function, the left arm is nearly normal, the right deltoid is wholly atrophic, but the trapezius and flexors of the right hand functionate. Some atrophy of pectorals and intercostals is evident. The severity of the case and the extreme condition at the close of the first seven months render the present recovery remarkable.

As some men prominent in orthopedics and neurology have condemned the use of electricity and massage, I have taken a recent article as illustrative of this side of the subject, *i.e.*, Dr. Henry Ling Taylor, professor of orthopedic surgery, New York Post-graduate School and Hospital; adjunct attending surgeon, Hospital for Ruptured and Crippled (Medical Record, October 15, 1910, page 660) says: "The conventional treatment by electricity and massage is completely ineffectual." To further support this

position, he continues: "This was publicly acknowledged by Dr. Bernard Sachs, of New York, a distinguished neurologist, etc., and chairman of the Collective Investigation Committee of the New York epidemic of 1907, at the Congress of American Physicians and Surgeons, at Washington," May 10, 1910. In these words he spoke of electricity and massage: "I consider that the time given to massage and electricity, in these cases, is time wasted. I cannot see that these same methods do any definite good."

It is true that Dr. Sachs made the above statement in May, 1910, as I was present at the time; but five months later, on October 24, 1910, at the New York Academy of Medicine, Dr. Sachs, in discussing this subject, spoke of electricity and massage in the highest terms, as he had previously done in the Journal of American Medical Association, October 22, 1910, page 1465, in the following words: "Electric treatment can hardly be overdone. It should be begun about one week after the subsidence of the febrile period and should be continued daily until full recovery . . . . or a chronic stage has been reached," *et seq.*, thus showing that a man of the highest standing, from a later and more comprehensive knowledge of the subject, completely changed his views of the value of electricity and massage.

That Dr. Taylor is unconsciously in accord is shown on the next page of his own article, where he speaks in the highest terms of vibration (which is a simple mechanical massage) and active and passive movements (which have always been classed under Swedish massage).

This case (Fig. 82) is shown to illustrate how massage and electricity, carried out by the parents alone, have won success, when many prominent physicians, who were consulted from time to time, made the darkest prognosis, with no promise of the use of either limb:—

Olive B., when 1½ years of age, had an attack of infantile paralysis, with both limbs involved; patient was not able to bear her weight on either limb for two years. During this time various physicians were consulted, and the mother was told that the case was hopeless. At the advice of an old physician, the mother herself

Fig. 82.—Success with massage and electricity.

purchased a battery and gave the child massage and electricity. At a later time, braces were ordered by institutions and applied, but as the mother thought that this did not aid in the recovery they were discarded.

Nevertheless, the mother proceeded in the massage and electricity "and manipulation to bring the foot around," keeping up the

treatment almost daily for twenty years. The girl, as I now present her, is 24 years of age; has no difference in the length of her legs, but the right foot is an inch and a half shorter than the left. She is just recovering from a corrective operation on this right foot.

In the Bulletin on Infantile Paralysis of the Massachusetts State Board of Health, 1909, Drs. Bradford, Lovett, Brackett, Thorndike, Soutter and Osgood, in speaking of the treatment, said:—

Electricity.—The different forms which may be used for this are the galvanic, faradic, static and high-frequency currents. In the early stages galvanism should be used on the nerve-trunks and faradism on the muscles, so long as their irritability for contraction is maintained. When the irritability of contraction to the faradic is lost, galvanism should be used, as having more influence on nutrition. With the returning muscle irritability, faradism should be used, and best by the use of the electrodes over the muscle points so as to obtain actual contraction of muscles rather than by the application of the electric current to broad surfaces. This serves as a distinct exercise to the muscle during its early stage of weak contraction. High-frequency and static electricity can both be used for their influence on nutrition rather than for their direct action on muscle contraction. It may be stated, in this connection, that the main dependence for actual results must be placed upon the galvanic and faradic currents.

REACTION OF DEGENERATION.

I believe much foolish stress has been laid upon the reaction of degeneration, and I wish to prove, from a large practical experience, how deceptive it may be.

It is said that a failure to obtain a muscle contraction by a galvanic or faradic current is an evidence of degeneration of the muscle fiber and that no improvement can be looked for in this paralyzed condition in the future.

Do we fail to obtain a contraction in most cases? No! We find that the cutaneous surface will not tolerate the pain of the current and we must desist before contraction

takes place, for most of our cases occur in children under 5 years of age, and they see no reason for enduring the electric pain. This is even true in other cases, when the age of the patient and his cutaneous tolerance are greater; the strong currents may give no reaction and still reappearance of function may occur.

Case B is shown to correct two long-established fallacies: One, that no improvement will occur whether spontaneous or under treatment after one year; many say six months. The other, that after failure to react to either the galvanic or faradic current, known as the reaction of degeneration, no improvement can be looked for.

Frank S., boy 16 years old; when 1½ years old had an attack of infantile paralysis involving his face and arm; fourteen years after this attack appeared at the Hospital for Deformities and Joint Diseases, hoping to receive treatment that would improve the condition of his face. He was turned over to Dr. Chas. Rosenheck, who had had five years' experience at the Roosevelt Hospital dispensary (department of neurological diseases) and also at the Harlem Hospital dispensary (neurological department), and who reported to me that he was satisfied that his reaction of degeneration was such as to be beyond all hope of the slightest benefit. I requested the other members of the staff to confirm this condition, as I wished to see if, after the lapse of fourteen years, with a positive reaction of degeneration, any improvement could take place. The improvement was so great after being under constant treatment for six months (the boy being able to close eye and produce wrinkles in his forehead, with a return to the normal outline of the affected side of his face) that he was shown at the Pediatric, Neurological, Orthopedic, and other sections of the Academy of Medicine. Many other similar cases have been encountered.

Hofer was able to demonstrate anatomically the misleading character of the reaction of degeneration in some cases. He cut down on muscles which had given the reaction of degeneration and found normal muscle fiber.

PRESENCE OF PAIN DURING ELECTRIC TREATMENTS.

The induction of pain during electric treatment is due to its improper use; the strength of the current should always be regulated so that contractions do not induce pain. When the parents stated that the treatment excites the child, the excitement was due not to the use but the abuse

Fig. 83.—Infantile paralysis at 1½ years of age; at 16 great improvement after six months' constant treatment. (N. Y. Hospital for Deformities and Joint Diseases.)

of the electricity. Continued overuse will result in a destruction of nerve-cells, a very detrimental effect, accompanied by a hysteric condition.

In conclusion, I wish to recall some salient points:—

1. Treatment should begin immediately after paralysis appears, but should be mild in the beginning.

2. The application of high frequency over the spinal column by its contracting action relieves the compression on the nerve-cells in the cord by the extravasated blood

and serum, and decreases the virulence of the toxin, inhibiting further extension of the process.

3. We obtain muscular contraction by the galvanic or faradic current, and thus prevent muscle atrophy during that period in which the muscles have not yet come again under the control of the will, and win a victory from what seems positive defeat. This I have proven in over 50 cases, who have had previous treatment from one to three years, in other institutions, without the ability to walk, or an ability to use their arms. Many of these cases have been shown at the section meetings of the Academy of Medicine, New York City.

4. The electric current should be the weakest that will produce a muscular contraction, and should not be continued in weak muscles when contraction ceases.

5. We should approximate the origin and insertion of muscles when applying electricity.

As a final summary of my own personal estimation of the relative value of electricity, active muscle education and massage, in the treatment of infantile paralysis, it is my opinion that the rating of good accomplished would be 55 per cent. from electricity in its various forms, 25 per cent. from muscle education combined with mental concentration on the physical effort, and 20 per cent. from muscle stimulation by massage.

CHAPTER XIV.

Physical Therapy, Massage, and Passive Motion.

PHYSICAL THERAPY.

Physical therapy is the reliance of the physician throughout the total period of treatment of paralytic cases of poliomyelitis. In cases not requiring corrective apparatus and intervention it is the chief treatment; in cases requiring orthopedic correction it has become more and more evident that the final success of the correction is dependent on supplementary untiring and intelligent physical therapy. Recent demonstrations of the value of physical therapy in the treatment of the chronic stage of infantile paralysis have contributed to the final recognition of long-ignored measures; we have noted with interest the acknowledgment from Dr. Hobart Amory Hare, of Philadelphia, to the effect that he now tells his students to use physical measures wherever possible, to the exclusion of drugs.

Hydrotherapy and electrotherapy have been considered elsewhere; we now come to the consideration of *massage*, *passive movements*, *resistance exercises*, and *muscle training and re-education*:—

I. The *massage* of a paretic muscle maintains nutrition by artificially stimulating the muscle cell, milking the venous blood from the part, inducing secondary hyperemia and the local elevation of a depressed temperature.

II. *Passive movements* maintain the normal range of motion of the joint, tending to prevent contraction; they are also the first step in muscle training and re-education.

III. *Resistance exercises.* Weakened muscles which yet retain some voluntary motion can be advantageously developed by discreet opposition of these movements by the operator (masseur). Mechanotherapy and the principle of the Zander machines are developments of the resistance exercise, and are of great value when power for operation by the patient is attained.

IV. *Muscle training and re-education* is called into play in that large class of cases in which the muscle sense is temporarily in abeyance due to a condition of suspended animation of the ganglionic neuron, or in which the motor function is taken over by uninvaded cells at another level of the cord.

MASSAGE.

I. Medical massage dates from the work of Dr. Metzger, of Amsterdam, and his followers, and his classification in a great measure still prevails.

1. We have the passive movements which are given to the patient by the operator.

2. Active movements made by the patient with the assistance or resistance of the operator.

The following are the manipulations:—

1. *Effleurage* consists of a centripetal stroking by means of the inner side of the thumb and first finger, the space of the hand intervening, milking and pressing the blood and lymph from the extremities toward the body.

2. *Frictions* are given with the thumb or the tips of fingers; they are strong, circular manipulations and are always followed by centripetal stroking.

3. *Petrissage* (kneading); this manipulation is performed by the tips of the thumb or the palm of the finger; it is used principally on the extremities.

4. *Tapotement* (percussion) is divided into five kinds:—

(*a*) Clapping, which is performed by the palm of the hand.

(*b*) Hacking, with the ulnar border of the hand.

(*c*) Punctation, with the tips of the fingers.

(*d*) Beating, with the clenched hand.

(*e*) Vibration, as the friction of the vibrator.

Zabludowski has shown that muscles regain their apti-

Fig. 84.—Massage and resistance exercises. (N. Y. Hospital for Deformities and Joint Diseases.)

tude for work much more quickly by a few minutes of massage than by rest for a longer time.

Dr. Benjamin Lee ("Hare System of Practical Therapeutics," vol. ii, page 321) states: "In the essential paralysis of infancy, truly wonderful results are obtained by massage."

The treatment should be entered upon the moment the acute inflammatory symptoms have disappeared and be

continued daily in the face of seeming absolute ineffectiveness. Cases in which no improvement can be detected for long periods often suddenly begin to improve and progress with great rapidity.

The effect of massage may be arranged as follows: Mechanical, reflex, thermal, electric.

1. The mechanical effects are by far the most important. They consist of the interchanging of cell contents under the influence of alternate pressure and relaxation; a quickened movement of the blood in the capillaries, especially in the muscular tissue; increased activity in the movement of the areolar fluid; acceleration of the currents of both blood and lymph in their respective channels.

2. The reflex or purely nervous effects of massage are obtained by light stroking and percussion. The former produces results which can only be explained on the supposition that it acts as a stimulant to the reflex system of nerves, the force used not being sufficient to account for any change on the mechanical theory.

3. The thermal effects of massage and movements are almost too apparent to need scientific demonstration; everyone is familiar with the fact that both muscular contraction in the form of ordinary exercise and simple friction develop bodily heat in a striking degree. Dr. Weir Mitchell, in his essay on "Fat, Blood, and How to Obtain Them," notes that he has frequently seen the strangely cold limbs of children suffering with infantile paralysis gain from 6° to 10° F. during massage.

4. The electric effect of massage results partly from the development of the surface heat, partly from the surface friction, partly from the attrition of the muscular fibers and cells, partly from the nerve stimulation and chemical action.

Most of the massage treatment given to affected chil-

dren consists of rubbing the skin, or moving the skin on the underlying fascia. To obtain the result desired and do the most efficient work, we grasp between the thumb and first finger all the tissue between the skin and bone, and by a process of pressing and milking, going from the extrem-

Fig. 85. Fig. 86.

Figs. 85 and 86.—Total paralysis of both legs. Treatment with massage, electrotherapy, muscle training. Perfect function. No atrophy. (N. Y. Hospital for Deformities and Joint Diseases.)

ity toward the body, the veins and lymphatics are emptied and elimination and assimilation improved. Deep kneading and brisk striking with the back of the first phalanx help to produce hyperemia. Vibration properly given is a good substitute for massage. By such an application of

thorough massage the local temperature of the skin is raised from one to three degrees.

Graham observes that muscles give a much more ready, vigorous and agreeable response to the will and to the faradic current after massage than they did before.

A child suffering from infantile paralysis was introduced; the affected limb having a surface temperature of 70° F., the poles of a battery were applied to a limb, and 11 milliampères were required to produce muscular contraction; the limb was then massaged and the temperature was found to have risen to 95° F.; the poles being applied at the same points, contractions followed the employment of only 5 milliampères. It is evident, therefore, that massage diminishes the resistance of the tissues to the electric current and increases the electric contractibility of the muscles.

An eminent authority recently said that in the treatment of infantile paralysis there was no virtue in drugs or electricity, and the only treatment that was beneficial was massage, and anyone could give that, *and he had it done by the child's maid.* Why should we be surprised at the growth of osteopathy and Christian Science, if such treatment is all we can offer to the parents of children affected with infantile paralysis?

The nerves in exposed areas, as the ulnar and musculospiral, the sciatic, peroneal and posterior tibial, should be protected from injury when giving deep massage. Undoubted cases of neuritis are not infrequent sequelæ of poliomyelitis, and may remain an incurable and lifelong affliction of the patient; we should see that they are never induced by trauma.

When the patient is receiving the combined treatment of massage, electrotherapy, and muscle training, ten minutes are sufficient time for each. I have seen several

cases of too prolonged massage treatment. To illustrate this I will cite 2 cases:—

L. H. Bethlehem: Child had a very severe attack of infantile paralysis, involving the extremities, back, abdomen and chest muscles, with total exhaustion and all muscles flaccid; he received massage for an hour and a half, or more, daily, with no improvement, but great exhaustion followed for three minutes after each application. The child has improved decidedly under more rational treatment by massage and electricity.

Case referred by Dr. Taggart, of Atlantic City: Child had involvement of left leg and arm. Treatment was given for one hour three times a day.

A strong, healthy man finds massage, which is applied over the whole body for an hour, a physical tax. What must three hours' treatment a day mean to muscles devitalized by the lost nerve-supply of infantile paralysis?

II. The period during which *passive movements* are of value is usually short; as soon as voluntary motion returns in the slightest degree it should be utilized until ingenuity is exhausted in devising means to such ends; according to Dr. John Ridlon, active movements performed by the patient himself have positive curative value probably greater than any other remedy.

However, there is a field for passive movement early in the paretic stage, while voluntary movement is inhibited by pain and fear as well as the paresis; passive motions are more easily accomplished when the patient is in the immersion bath, whose warmth and buoyancy permit a range of movement painful elsewhere. Passive motion assists in maintaining the range of movement of the joint, in that it exercises the paralyzed muscle, and briefly inhibits the overaction of the unopposed healthy group.

Passive movements performed slowly several times in sequence, and then attempted by the patient, are the first step in muscle re-education.

CHAPTER XV.

Therapeutic Exercises Performed Before a Mirror.

"What the mind conceives the body achieves."

THE purpose of this chapter is to call attention to a method of conducting therapeutic exercises before a mirror during which the muscle effort is directed by mental concentration on the act.

The physiologic entity of man consists of a muscular system, a neural system, and a life-giving function maintained by the viscera,—lungs, heart, stomach, pancreas, liver, spleen, kidneys, intestines, etc.,—which require exercise for proper adjustment and functioning. It is known that muscular exercise profoundly affects the functions of respiration, circulation, nutrition and excretion, while the heat-controlling mechanisms of the skin and sweat-glands may be stimulated thereby to greater activity. Hence, in diseases of various parts of the body much can be accomplished by selecting exercises for such function or organ as will tend to re-establish its normal relation to the economy.

It is also well to entertain the fact that mental concentration has a stimulating effect on growth, whether in intellectual brain development accomplished by the study of higher mathematics, or resulting from the concentration of effort required for voluntary muscle contraction attained by gazing at the reflection of the particular muscle or muscle group in a mirror. The relative power of mental concentration is the accepted measure of the superiority of one mind over another. A moment's thought makes clear

the fact that the nerve control and nerve efficiency displayed by the Oriental muscle dancer can be developed by any person in any set of voluntary muscles, if a proper

Fig. 87.—In mirror X is seen foot placed at right angle to be brought up through arc of 30 degrees. In mirror Z observe instructor's hands under leg and foot. (N. Y. Hospital for Deformities and Joint Diseases.)

effort is made for development of sufficient nerve force and nerve control.

We must now realize that the concentration of the mind on the muscular effort not only initiates the movement, but determines blood to the controlling nerve-centers,

Fig. 88.—Normal standing position, showing deflection of spine before mental and muscle effort for correction. (N. Y. Hospital for Deformities and Joint Diseases.)

Fig. 89.—Deflection corrected. Maximum muscle effort at point of pencil. (N. Y. Hospital for Deformities and Joint Diseases.)

producing growth and development in the conducting nerve-trunk to its most distant filament. It was demonstrated by Anderson, of Yale, and others that when a person is securely placed on a body balance, and concentrates his mind on one extremity, the balance tips in the

Fig. 90.—Plumb-line test to demonstrate muscle contraction. (N. Y. Hospital for Deformities and Joint Diseases.)

direction of this limb, showing that a hyperemia, a true determination of blood to the part, had been secured.

It has been found, in post-mortem examinations of the human brain, that when motion of an extremity is guided by mental concentration the convolutions of the gray matter of the brain presiding over this motor area are increased; the reverse of this process has been frequently

demonstrated: post-mortem examination of the cerebral cortex of an individual minus an extremity from intra-uterine amputation demonstrates lack of development of the cortical center for that area.

Other conditions being equalized, that is, the securing

Fig. 91.—Plumb-line test to demonstrate muscle control. Note approximation of waist to plumb line and increased bulk of left erector spinæ. (N. Y. Hospital for Deformities and Joint Diseases.)

of ample nourishment and the absence of undue fatigue, the stimulus transmitted from the brain to the periphery depends on the calibration of the conducting nerves, as the diameter of copper wire regulates the volume of electric current.

Normal and equalized conditions are not, however, found in the nerve-trunks after invasion by an attack of

poliomyelitis. "The anterior roots at the (affected) level are decreased in size. A similar condition exists in the motor nerves" (Vulpius). The trophic function has been seriously impaired, and there is a constant condition of hyperexcitability which produces undue fatigue. In addi-

Fig. 92.—Muscle effort and muscle balance (erector spinæ) acquired by corrective exercises for lateral curvature. Three-year student at leading school of physical culture. Unaware of lateral curvature; subsequently absolutely corrected by Frauenthal method. (N. Y. Hospital for Deformities and Joint Diseases.)

tion some filaments have suffered degeneration and atrophy subsequent to the destruction of their peripheral neuron, the motor cell.

The problem of the mirror work to be described is, then, not the simple problem of securing a certain number

of contractions daily of an unused and paretic muscle. To achieve a good end-result we must produce:—

1. Muscular contractions to (*a*) prevent the atrophy of disuse; (*b*) promote regeneration.

2. Determination of blood for nourishment to secure trophic repair and growth for (*a*) impaired nerve-trunks;

Fig. 93.—Therapeutic exercise before a mirror. Individual instruction. (N. Y. Hospital for Deformities and Joint Diseases.)

(*b*) paretic muscles; (*c*) inhibited structural growth, cartilage, and bone.

3. Re-establishment of sensory and motor impulse to (*a*) directly increase calibration of nerve; (*b*) co-ordinate nerve impulse now wasted.

4. New anastomotic association paths for impulses whose motor tract or level has been seriously invaded or destroyed.

The effectiveness of the following method outlined has been abundantly demonstrated by the results we have shown from time to time with cases of poliomyelitis and locomotor ataxia in various clinics held during the past twenty years:—

METHOD OF MIRROR TREATMENT.

Treatment should be given before a large and well-lighted mirror, so that the patient may see all parts of his body clearly. There should be no clothing in use which hampers movement or obscures the view. The patient should be in stockingfeet, or soft moccasins. We do not sufficiently appreciate what a foot can do when untrammeled.

In corrections of the torso, particularly lateral curvatures, a compound mirror is needed, and should be so arranged that the patient can constantly and without effort observe the erection of the spinal and other muscles of the back.

The mirror should extend to the floor; in all foot work, and most of the leg work, and also in the stretching and rising for lateral curvature, the child's vision would be at once obscured by a mirror hung above the floor line. While some of this work can be done on an absolutely steady table drawn close to the mirror, a rug thrown on the floor provides a base of operations for the patient whose stability is assured, and one factor of distracted attention is thus eliminated.

The most important factor in the treatment is the instructor. A trained instructor with a high grade of intelligence is needed for this work. The graduate in physical culture needs a postgraduate training of at least one year to become proficient, a good knowledge of anatomy, tact, patience, and the personality which insures

obedience. The graduate in physical culture who has acquired muscle control by persistent training is the best instructor.

There should be nothing in the range of vision to distract the attention of the patient from the work in hand. In private practice at office or home, no one should

Fig. 94.—Therapeutic exercise before a mirror. Individual instruction. (N. Y. Hospital for Deformities and Joint Diseases.)

be present in the room besides the instructor and the patient. Individual treatment is desirable, for by this means we have obtained the best results. However, in institutional work, children over 3 years of age are given instruction in groups of 6 or more. We have such daily classes at the Hospital for Deformities and Joint Diseases. The classes have been visited by the leading neurologists and pediatricians of New York City, and the

visitors unite in wondering and favorably comment on the ability of the children for concentrated work, which is continued with total unconsciousness of the presence of strangers.

Concentration, directed by an able instructor, is the keynote of this treatment, to which the mirror forms a most valuable aid. The patient's whole attention must be centered on the part under treatment, and when possible on the particular muscles involved. When the child observes the desired action taking place in the mirror, and realizes that his effort is bearing fruit after a few days of work, it is surprising the excellent effort he will put forth.

In the beginning, light massage or beating of the muscle will aid its action. This is also attained by approximating the origin and insertion of the muscle, which increases the belly bulk, with a corresponding increase in contractile force. The instructor must make clear to the patient the muscles to be brought into use. The contraction should be made slowly for maximum effort and effect.

When the contractile force is not sufficient to move the limb, the instructor aids in the desired motion, at the same time compelling the patient to make all mental effort toward its attainment. As the muscle becomes stronger, the needed assistance is lessened. The muscle is allowed to take up more of the work, run the whole of the exercise, and receive the maximum amount of work possible in its weakened condition. The *exercise must always stop short of the fatigue point. Fatigue, if encountered, will be both mental and physical and partake of the nature of neurasthenia.* One must be guided by judgment and experience as to the amount of exercise to be used, particularly during the first instruction with seriously impaired muscles. It is here again that the services of a competent instructor are

invaluable for the welfare of the patient and the results the physician desires to attain.

The muscles most frequently involved in infantile paralysis are those making up the perineal group. Placing the child on a chair in a comfortable position before the mirror,

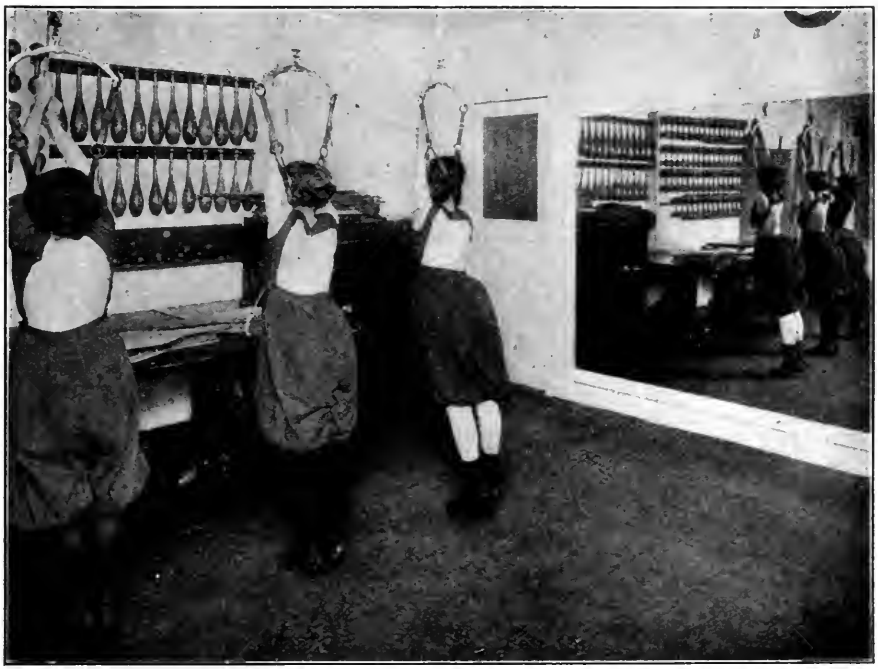


Fig. 95.—Suspension correction for lateral curvature. (N. Y. Hospital for Deformities and Joint Diseases.)

the instructor approximates the origin and insertion of the muscles composing the group by bringing the foot up to a right angle with the leg; he then urges the child to aid in bringing up the small toes at the side of the foot through an arc of about 30 degrees. If the child cannot do this alone, the instructor places one hand on the knee to keep the leg in position, and the other hand under the foot; this greatly aids the child's effort to make the required con-

traction. This should be repeated several times, but never to the fatigue point. Each set of muscles should be contracted in a similar manner. If the motion cannot be brought about, still the mental effort should be made for this attainment. As a result of using this method I have been able to show at medical clinics 43 ambulant patients who were formerly unable to walk for a period of from nine months to four years. Many of these cases had been referred to the institution by Drs. Kerley, Koplik, Mandl, and other men of standing.

In the treatment of lateral curvature due to infantile paralysis (and this is the primary cause of a majority of mild as well as very deforming cases of curvature) we are handling paretic or paralyzed muscles on one side of the spine and unopposed, overcontracted, healthy muscles on the opposite side. Securing a permanent result is dependent on the success in equalizing this muscle force, or in *re-establishing in the weaker muscles a strength equivalent to that on the opposite side.* The correction effected by Abbott's overcorrection method is promptly lost on removal of the jacket unless this muscle re-education and actual renewal is attained. Abbott himself now follows up the overcorrection with muscle education.

I have seen so many cases of return of function to paralyzed extremities by the persistent use of this line of treatment that I would advise the discouraged to renew effort, expecting to meet with such agreeable surprise as I have at the results accomplished by the work.

Arthur H. after initial attack received four months' treatment at one clinic, eight months' treatment at another clinic, and on going to the third clinic an operation for fixing the foot and ankle was suggested, as apparently there was extensive atrophy and no function.

At that time she (Mrs. H.) met the mother of a patient who had had her child for treatment at the second institution for three

years, with no ability to stand or walk, and Mrs. H. was informed by her that under treatment at the Hospital for Deformities and Joint Diseases for five months the child was able to walk without braces. Mrs. H., regarding this as an absurdity, made a special

Fig. 96.—Able to walk with almost normal gait after one year's treatment. (N. Y. Hospital for Deformities and Joint Diseases.)

visit to the child's home, and, seeing what she regarded as a miracle, brought her child.

This child was under treatment for seven months before he gained any promise of bearing his weight on his right leg, and after one year he is able to walk with almost normal gait.

BREATHING EXERCISES.

The alarming increase in the respiration rate during the acute stage of poliomyelitis is evidence that practically every case suffers from some involvement of the respiration center. The fatal cases are usually if not always terminated by paralysis of the respiratory tract. Every case of upper-extremity involvement presents some atrophy of the serrati; the serrati are involved in all cases of postparalytic scoliosis. The majority of these children, whether left with a lesion of upper or lower extremity, present a very indifferent chest expansion; this is so noticeable as to suggest that the so-called phthisical chest is the inheritance not from a tuberculous ancestor, but from one who had had an inhibiting attack of poliomyelitis.

To overcome this serious defect, as well as to provide oxygenation for all the body tissues, breathing exercises should be made a constant accompaniment to any and all physical therapy undertaken.

Resistance movements and *muscle training* are of most value in treatment. The earlier they can be begun after the subsidence of all irritation, the better the outcome.

It has been the attention of this kind that has yielded results after operation: where the work of exercise has produced *most* of the improvement and *not* the operation.

We have found that there is invariably some regression of the original paralysis, and the best results therefore naturally seem to be obtained from the treatment of cases referred early in the course of the disease. We believe that the best results of treatment do result from treatment instituted as early as possible and judiciously given; we have, however, had complete restoration of function in cases referred to us as hopeless and with complete loss of function as late as eight months after the acute stage.

In several cases between 12 and 18 years of age in which the patient could not hold any article in the grasp of the affected hand, and no improvement had been noted for periods of time extending to six years, by a careful system of training and development of the muscles the hand was enabled to grasp and use a knife or fork, and finer movements were gradually acquired until the patient could write with the affected hand.

In this treatment, time should not be considered a factor, either in a hopeless prognosis or in the discouragement which causes the parent to stop treatments. Failure is usually the result of neglect; it may be due to faulty methods of the use of electricity in its many manifestations, to overmassage or injudicious exercise.

The apathetic and awkward child will develop enthusiasm in the hands of a skillful and tactful instructor, fresh association paths for muscle impulse will develop, co-ordination between action and impulse will be gradually restored, while at the same time deformities arising from the overaction of unopposed muscles must be recognized and inhibited.

It has been said that association fibers in the decussation tract of the brain are able to take on the function of fibers whose cortical relations have been impaired. If this is true of the higher centers, we are justified in expecting a similar adjustment of transmission of impulse in the various levels of the cord. Observation demonstrates that such renewal of association paths occurs.

CHAPTER XVI.

Mechanotherapy.

MECHANICAL apparatus was first devised for the bed-fast paralytic with muscles atrophied from the waist down, to enable him to walk. The iliopsoas muscle, a great

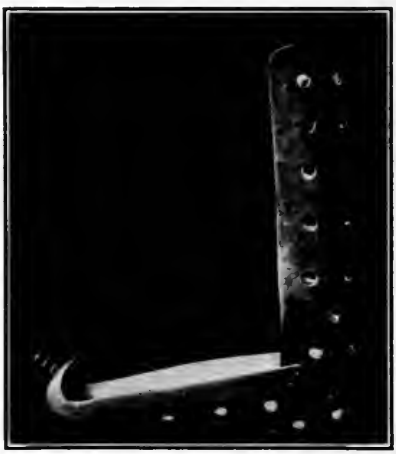


Fig. 97.—Night support for foot and instep. Aluminum. Weight, 3½ drams (N. Y. Hospital for Deformities and Joint Diseases.)

levator of the thigh, takes its origin and innervation in part from the dorsal spine; it is therefore found, in numerous cases of paraplegia, that the iliopsoas is wholly unaffected, and may be utilized in locomotion, once the flaccid limbs are stiffened and properly supported. Heine constructed a primitive brace in 1840, which stiffened the legs of a paralytic, and took its support from a metal belt encircling the rim of the pelvis; this brace was successful and a drawing of it was published. The clever mechanism stimulated the production of apparatus with more delicate

Fig. 98.

Fig. 99.

Figs. 98 and 99.—Private patient at the hospital with infantile paralysis involving muscles of both legs and spine. Able to get about by proper-fitting braces. Fig. 99 shows anterior view. (N. Y. Hospital for Deformities and Joint Diseases.)

adjustment and adaptation, until today in England and Germany it is possible to secure a glove-fitting support of molded leather with a feather-weight framework of hollow steel.

It has been found that splints and braces, when used understandingly, are of value in the treatment of infantile

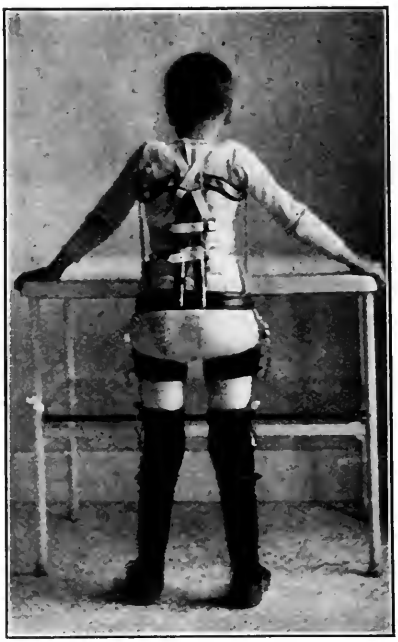


Fig. 100.—Same as Fig. 98, posterior view.

paralysis from the time of onset. Their abuse in the hands of the unskillful and negligent is very detrimental to the patient.

I wish to state with emphasis that any type of apparatus that is bandaged to the limb, or is attached with straps and buckles, is detrimental to the paralyzed muscles. An atrophy of muscle is produced, either throughout the muscle length or locally. Where the collar of the brace

Fig. 101.

Fig 102.

Figs. 101 and 102.—Neglected case of paralysis of both lower extremities with involvement of muscles of eye (abducens and left oblique). Boy had not walked until splints were applied. He is now improving under treatment with electrotherapy, massage, and muscle training. Fig. 102 shows brace; motion confined to one axis.

rests will be found an indentation of the muscle substance, due to pressure atrophy, which can be plainly palpated.

Another prevailing evil is the encasing of these paralyzed muscles in plaster-of-Paris or starch bandages, thus adding confinement and pressure atrophy to the already

Fig. 103.—Hyperextension of knees. (N. Y. Hospital for Deformities and Joint Diseases.)

damaged musculature, when every effort should be made to retain the tone of the muscle until it again comes under the control of the will and renews its function. More damage can be done with this type of confinement than can be regained by the muscles in a year's treatment and with our best endeavor.

Another grave objection to braces is that parents depend on the brace and neglect treatment.

No appliance should be used that is not removed

Fig. 104.—Same as Fig. 103, with hyperextension of knees corrected, anterior view.

Fig. 105.—Same as Fig. 104, lateral view.

daily to permit the treatment outlined in the chapters on physical therapy.

With this limitation distinctly understood, indications and methods for treatment by mechanotherapy are given.

The orthopedic splint or brace is used in infantile paral-

ysis as a (1) prophylactic, (2) supportive, and (3) corrective measure.

1. In the early stage during onset and regression of paralysis, as a prophylactic support and protection from dragging of the bed-coverings or faulty posture.

Fig. 106.—Paraplegia, with slight spontaneous improvement of muscles of left leg. (N. Y. Hospital for Deformities and Joint Diseases.)

2. During the first months of convalescence to maintain function and nutrition, to inhibit contractures, to lessen muscular tension, to maintain muscular balance, to maintain the integrity of ligaments, muscle tendons, and joint capsules.

3. In the later periods of the disease, mechanisms are

used for the fixation and limitation of mobility of flail joints and flaccid extremities, for muscle substitution, and to correct paralytic deformity.

Splints.—As the acute constitutional symptoms of the

Fig. 107.

Fig. 108.

Figs. 107 and 108.—Same as Fig. 106 after three months' treatment.

attack subside a lingering neuritis will force the patient to assume the most comfortable position, and one which favors the unopposed action of the unaffected muscles. Such posture, by favoring contractures, conduces to the development of contractures and deformity. This is particularly true of the child or youth still in the developmental

period, whose tissues and bones may be considered malleable. Thus, a girl of 15 years of age, in the fourth week of an attack of acute poliomyelitis (previously diagnosed and treated as rheumatic fever), was found with marked

Fig. 109.--Old operation and brace treatment. Uncured. Note pressure atrophy from brace. Paralysis of both lower extremities. Overtreated to inhibition. Final recovery on moderate treatment.

contracture of leg on thigh, and a footdrop, both of which conditions might have been lessened or prevented by light splinting.

The pressure of bed-clothes on the paretic foot and ankle is obviated by the use of a light aluminum splint applied to the heel and plantar surface of the foot, and

projecting an inch or more beyond the great toe. The use of a bed-cradle is directed in the chapter on treatment.

All mechanisms which are used in the early stage of the disease to inhibit contractures and deformity should

Fig. 110.—P. S., scoliosis following acute poliomyelitis, erect. (N. Y. Hospital for Deformities and Joint Diseases.)

be devised to interfere as little as possible with circulation or nutrition. Plaster-of-Paris bandages encircling the trunk or extremities and splints tightly secured by a bandage are *not to be used.*

Supportive Apparatus.—Mechanisms which aid locomotion and activity are of much importance. When no

deformity exists the loss of function from paralyzed muscles can be supplemented by mechanical contrivances which make the bones and the apparatus unite in supporting the body weight. The exercise thus obtainable develops

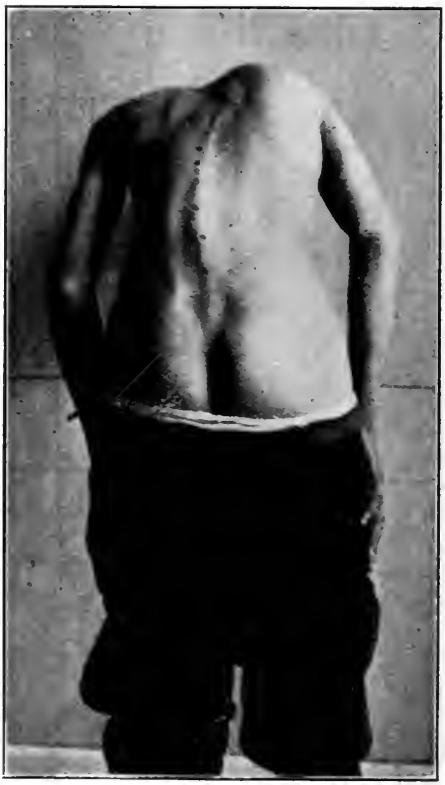


Fig. 111.—Same as Fig. 110, stooping.

the weakened and paretic muscles, resulting in a restoration of the motor function.

The simplest mechanism is a walking chair.

In many of these cases, by a proper application of therapeutic and mechanical agencies, we can circumvent subsequent deformity. The brace, however, must not be too heavy, and the adjustment must be carefully regulated.

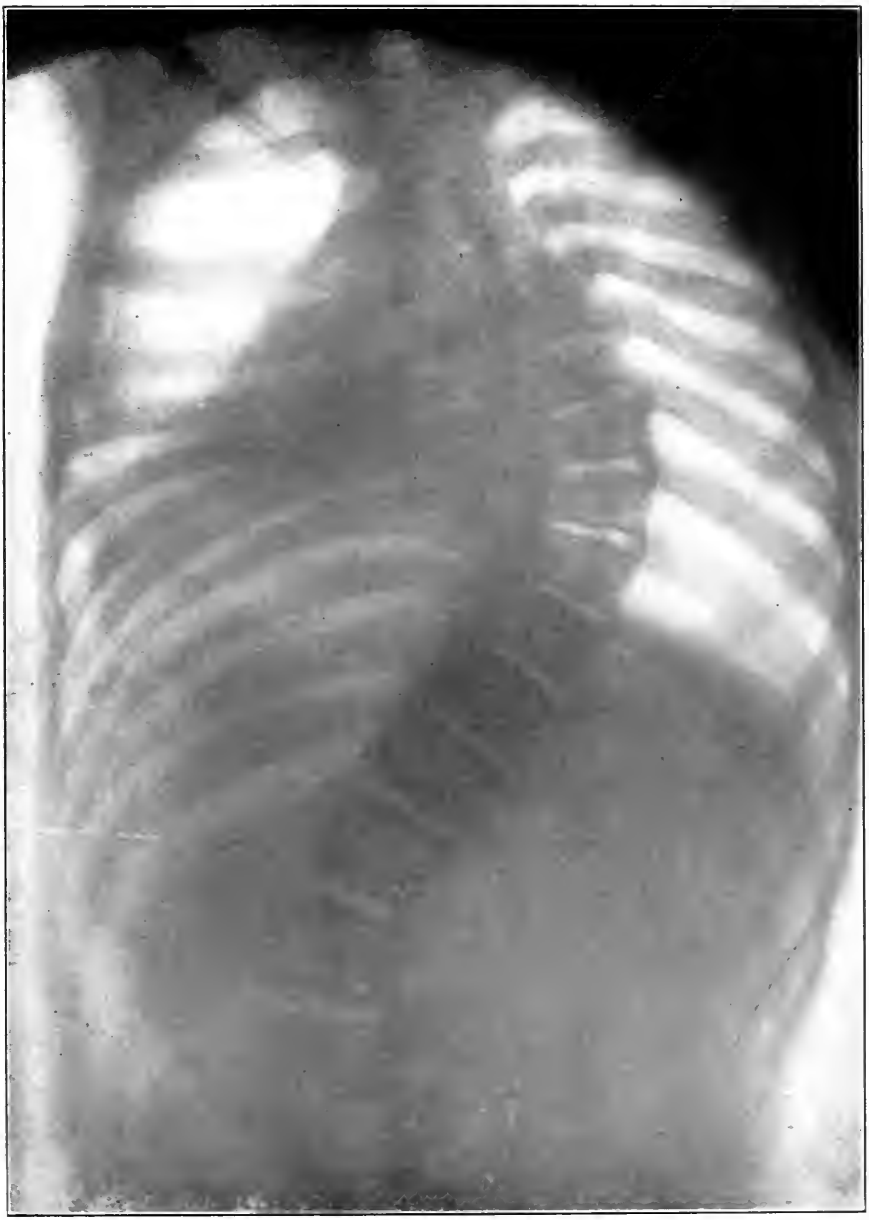


Fig. 112.—X-ray of spine of P. S.

Jones, of Liverpool, states that "nothing has tended more to the discredit of the practice of orthopedics than the lumbersome and complicated machinery with which surgeons have loaded their unfortunate patients." It fre-

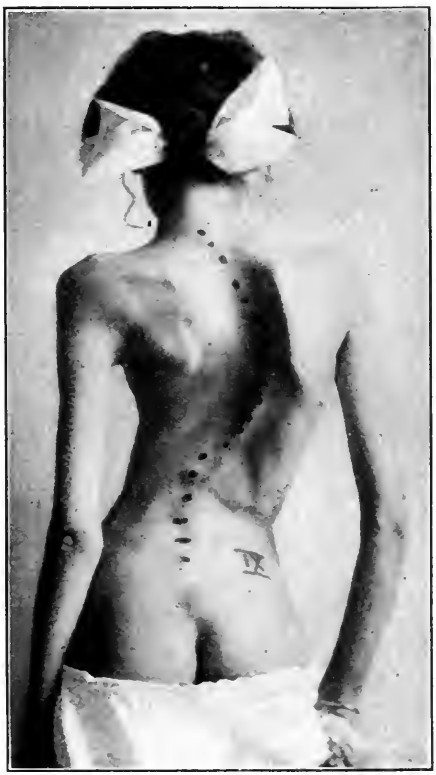


Fig. 113.—M. K., scoliosis following acute poliomyelitis, erect. (N. Y. Hospital for Deformities and Joint Diseases.)

quently happens that parents realize this and discard braces, which not infrequently are found to weigh from one-twelfth to one-eighth of the weight of the child. I have a large scrap-heap of such useless splints.

In the chronic stage of poliomyelitis apparatus is used to fix the flail joint and limit its motion to one axis only,

to control the degree of motion in that axis, to replace paralyzed muscles, and to correct deformities.

The cheapest form of splint is the malleable-iron rod bent to fit the part, and attached to the body with leather

Fig. 114.—Same as Fig. 113, stooping.

and steel bands. The more intricate modification of this apparatus, made of steel, is provided with movable lock joints, which may be fixed or released at will, which are further controlled with a front and back stop, the joint being uniaxial, or a gimbal joint which permits lateral movement; this apparatus also takes its fixed position from

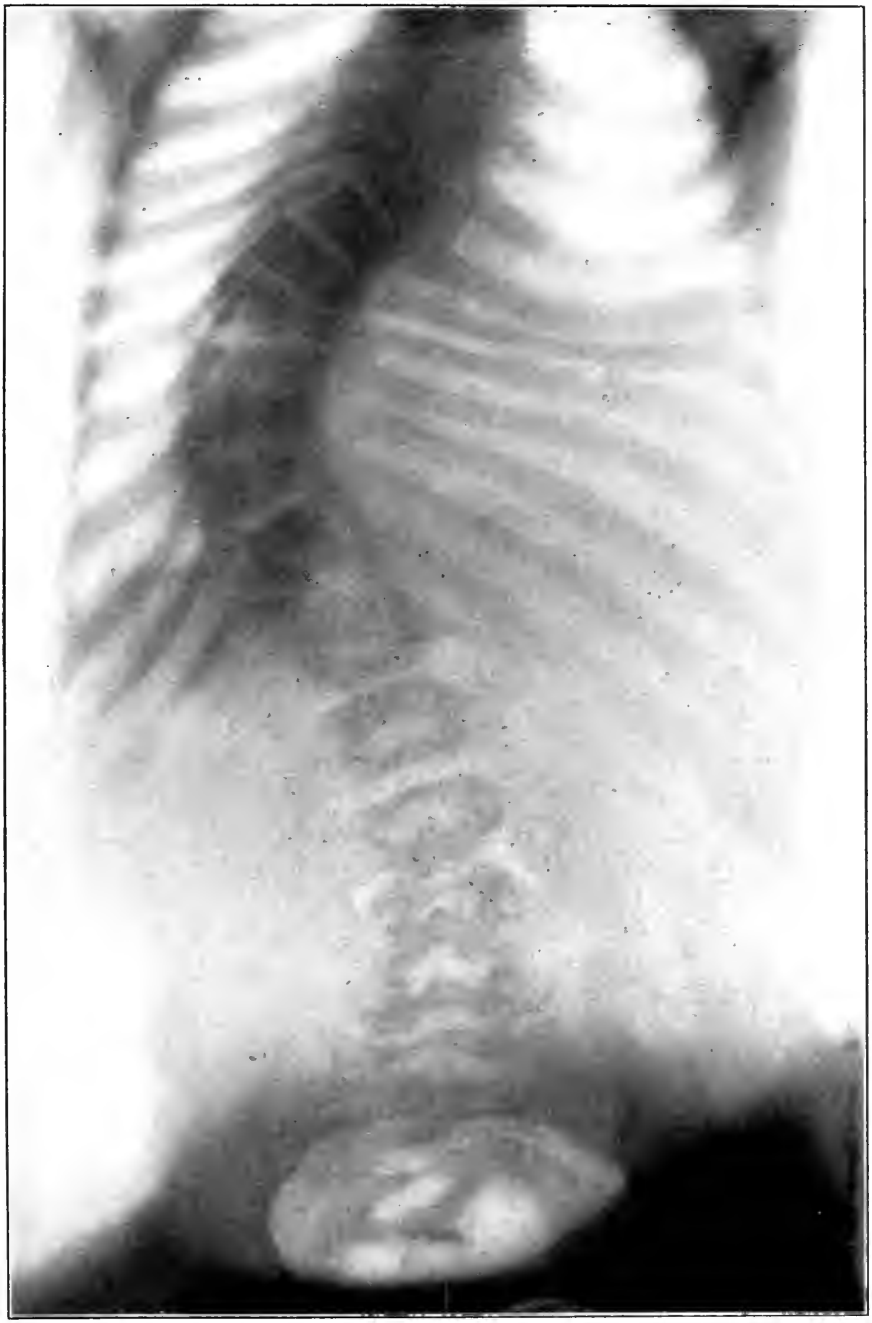
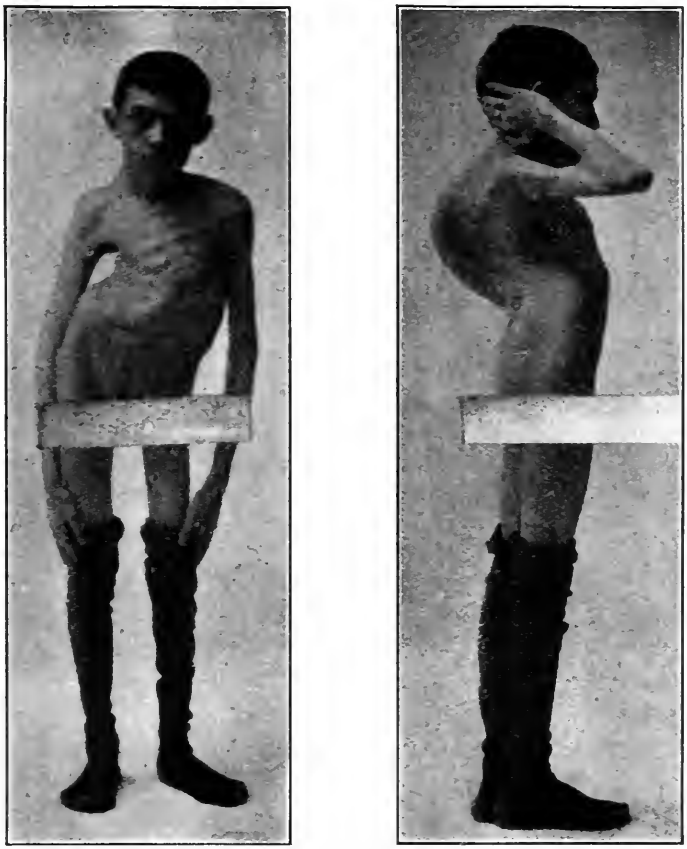


Fig. 115.—X-ray of spine of M. K.

straps and bands which encircle the body and extremity. When the use of apparatus is to be temporary only, and for one of the working class, these braces are sufficient.

Fig. 116. Fig. 117.



Figs. 116 and 117.—Extreme kyphosis following acute poliomyelitis. (N. Y. Hospital for Deformities and Joint Diseases.)

Made of the lightest construction the material admits, readjusted at frequent intervals, removed daily for the treatment necessary to offset pressure atrophy and the interference with circulation, they are indispensable for

some otherwise helpless cases. The pressure of the encircling band has been avoided in an apparatus constructed by skilled artisans in England and Germany in which the pressure is evenly distributed over the limb or torso by

Fig. 118.—Same as Fig. 116.

Fig. 119.—Same as Fig. 116.

the use of molded leather sheaths. (These are known by the name of the first manufacturer as braces.—Hessing.) A cast of the extremity is made, and to this the wet leather is molded and left a considerable period of time; in the mean time a very light supportive framework is con-

structed of hollow steel rules, whose construction and mechanism are exactly adapted to the needs of the wearer. The framework and its sheathing are now united and form ambulatory splint of such nice adaptation to the patient and his needs as to be wholly concealed by a shoe and stocking. This apparatus is expensive in preparation, and

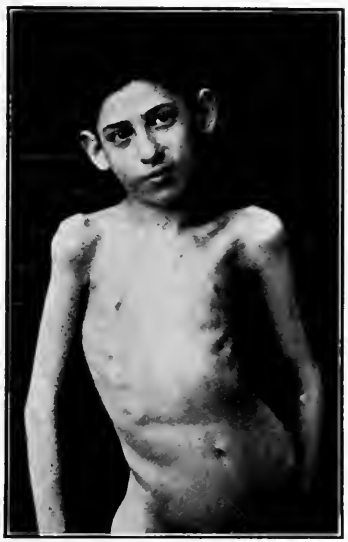


Fig. 120.—Scoliosis. (N. Y. Hospital for Deformities and Joint Diseases.)

in maintenance, for the parts must be renewed, the leather wears through, splits from exposure to moisture, etc. Yet there are many parents in this country who greatly desire to save the tender-hearted child from the unpleasant publicity of visible apparatus, who would gladly purchase such apparatus, and renew it so long as it was needed. There is an opportunity now for a clever artisan and mechanic to build up a business in this line in every large city. It is a matter of regret that such apparatus at present cannot be obtained in America.

Paul D., acute illness, July, 1910. Referred by Dr. Chas. G. Kerley, Oct. 16, 1910. Removed to hospital. Paraplegia, with slight spontaneous improvement of muscles of left leg. January, 1911, after three months' treatment the boy is able to stand alone in the lock-joint braces, is able to lift both legs to an equal or right angle, and to bear additional weight of braces.

Fig. 121.—Scoliosis with incorrect application of jacket. (N. Y. Hospital for Deformities and Joint Diseases.)

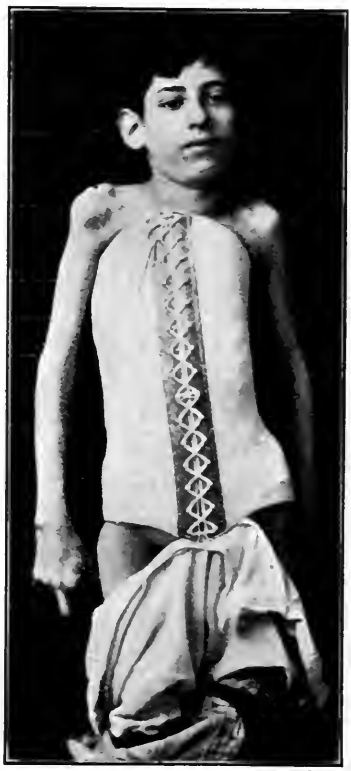


Fig. 122.—Scoliosis with correct application of jacket. (N. Y. Hospital for Deformities and Joint Diseases.)

The molded leather sheath is, however, open to the same objection, although in a much lessened degree, which holds in regard to the use of the band brace. Vulpius, who has had an opportunity to make use of and observe results

from these supports, states, in his work on infantile paralysis: "The atrophy which results from the long wearing of an apparatus is much in evidence with the band type of

Fig. 123.—Hammock suspension sling for forcible overcorrection of scoliosis while applying plaster jacket. (N. Y. Hospital for Deformities and Joint Diseases.)

fixation, but can also be demonstrated with the sheath type, for after some months the appliance becomes too large, and no longer fits the limb closely. The subcutaneous fat is the first to go, followed by the muscles, and finally by the bones, as the Roentgen rays have shown us."

The Substitute Muscle or the Elastic Tractor.—Spiral steel springs and india-rubber bands have been utilized with great ingenuity to replace paralytic muscles. Thus an artificial quadriceps has been arranged of two strips of stout gartering which cross the knee obliquely and are attached to the leg splints right and left above the knee,

Fig. 124.—Plaster jackets for scoliosis applied in attitude of overcorrection, and fenestrated for development of atrophied muscle. (N. Y. Hospital for Deformities and Joint Diseases.)

and below it; the crossing is further articulated to a semi-lunar band of steel which is hasped to the artificial knee-joint right and left. The tensile strength of the elastic is sufficient to overcome and oppose the flexors of the knee, or, at any rate, "to pull the affected limb into the extended position as soon as the flexors cease to act" (Volkman). Similarly an artificial gastrocnemius of spiral springs takes

its origin from the back of the leg band and is inserted into the shoe just above the back of the heel.

Mechanisms for Contractures.—Mechanisms for the prevention of deformity are still more intricate and de-

Fig. 125.—Plaster jackets for scoliosis applied in overcorrection, and fenestrated for development of atrophied muscle and compressed lung. (N. Y. Hospital for Deformities and Joint Diseases.)

pend upon the principle of continuous mechanical traction for effective results; the treatment is tedious, expensive, and uncertain in results, and it is questionable if this treatment alone would successfully prevent or correct existing contractures.

THE ANKLE-JOINT.

The most frequent defect that interferes with locomotion is paralysis of the peroneal group, and muscles about the foot and ankle-joint. In mechanical correction it must be determined whether the brace should be directly coapted with the foot, or attached to the shoe. A brace complete in itself, with foot-plate in apposition, will permit better opportunity to adjust mechanical ankle-joint, and may be worn with shoe. It is almost impossible for the artificial ankle-joint to be properly adjusted when the brace is attached to the shoe.

CHAPTER XVII.

Surgical Treatment of Poliomyelitis.

Although many cases in the chronic stage of infantile paralysis arrive at a condition where surgical measures are desirable, our attitude toward surgical interference is always a conservative one. We have found that some of the best results we have had have been obtained in cases which have discontinued attendance at other clinics because operation was insisted on, and in which our end-results were attained without recourse to surgery. The longer operative procedure is deferred, the better the permanent result will be, and in operations about joints one is less liable to interfere with the epiphysis of long bones and thereby defeat growth. The consensus of the best men is that ankylosis of knee and ankle should never be done before 12 years of age. Many operations for tendon transplantation and joint stiffening are done which would not be begun if the true spirit of the eleventh commandment was observed: nor would the surgeon do them on his own child, for by patient care, non-surgical treatment, and attention an end-result much more gratifying would be obtained.

Many cases in the chronic stage of poliomyelitis, owing to (*a*) lack of treatment, (*b*) improper treatment, (*c*) faulty application of braces, (*d*) sole dependence on brace, arrive at a condition where surgical measures are necessary.

The operative procedures are:—

1. Tendon lengthening.
2. Tendon shortening.
3. Tendon and muscle transference.

4. The application of artificial tendons and ligaments.
5. Joint stiffening; arthrodesis.
6. Nerve transference and nerve grafting.

It has been observed by Jones, of Liverpool, and other orthopedic surgeons that, contrary to general expectation, paralyzed limbs are most tolerant of operative interference; wounds heal well and quickly, and suppuration is less prone to occur than after operation on healthy limbs. Inasmuch as the tissues of these limbs have lost wholly or in part their trophic control, it is interesting that this fact has been observed and recorded by operators.

1. TENDON LENGTHENING.

The operation of subcutaneous tenotomy dates from the work of Stromeyer in 1831. This is a simple and safe operation for the correction of deformity, if one is familiar with the relation of the nerves and arteries; when such relations are not clearly in mind, one should do an open operation; the open method is also the operation of choice when the hamstring muscles are the point of attack.

Resection of the tendo achillis. The peroneal group of muscles is, perhaps, the most frequently involved of all muscle groups, and the paralysis is followed by drop-foot (talipes equinus), which is augmented by the unopposed action of the posterior group of muscles terminating in the tendo achillis. The cutting of this tendon is a very simple operation.

Technique of Operation (Tenotomy of Tendo Achillis).—The previously sterilized skin surface is painted with tincture of iodine, 3 per cent. solution. A small opening is made through the skin one inch or one and one-half inches above the os calcis; into this opening a blunt-pointed tenotome is thrust, the blade facing toward the skin, with the tendon between the blade and skin; the foot, flexed

sharply on the ankle, holds the tendon taut, while by a gradual sawing motion the tendon is cut through. When applying the dressings care should be taken to avoid inserting any dressing between the two separate ends of the tendon, which would produce non-union. The wound is sealed with surgeons' plaster and the foot flexed on the

Fig. 126.—Old talipes with atrophy. Reduced in one sitting. (N. Y. Hospital for Deformities and Joint Diseases.)

leg in an overcorrected position, and encased in plaster bandages.

The regenerated tendon is much stronger if the full separation is done immediately at the time of its severance. This fact was first demonstrated by Young, of Philadelphia, in operations on rabbits. English operators prefer to wait until partial healing takes place, when they subject the tendon to stretching, after which healing is allowed to proceed.

In operations at the Hospital for Treatment of Deformities and Joint Diseases the tendon is always severed and separated, and the foot overcorrected. In no case have we failed to get union.

2. TENDON SHORTENING.

It is the rule in infantile paralysis to find the weakened and paretic muscles overstretched, by the overaction of the unopposed, unaffected, antagonistic group; in place of lengthening the tendons of the strongly contracted healthy muscles, it is often desirable to shorten the stretched tendon of the relaxed and paretic group. "Taking in the slack" is accomplished by freeing the tendon, folding the tendon on itself, and sewing the folds together with silk. Wound infection is to be avoided with the usual surgical technique, the wound closed with surgeons' plaster, and the limb retained in overcorrection until healing has taken place, and for a number of weeks subsequent to the healing. The retention dressing should be removed for massage and electric treatment. Paralyzed muscles can often be restored to usefulness by shortening their tendons, or by the combined operation in which tenotomy is done on the spastic antagonistic group. Muscle groups which seemed almost wholly degenerated and were badly stretched have resumed motor functioning when the slack was taken up in this manner.

3. MUSCLE AND TENDON TRANSFERENCE.

In some cases of partial paralysis muscular balance is re-established by the transplantation of the tendon of a sound muscle to a new point of leverage which enables it to take over the function of the paralyzed muscle group. The operation originated with Nicoladoni in 1882, who

operated on a patient having talipes calcaneus and successfully corrected the deformity by attaching the peronei to the tendo achillis. Parish and Drobnik utilized a similar method in other forms of club-foot in 1892, and were followed by Winkelmann, Vulpius, Goldthwait, Long, and Schrantz.

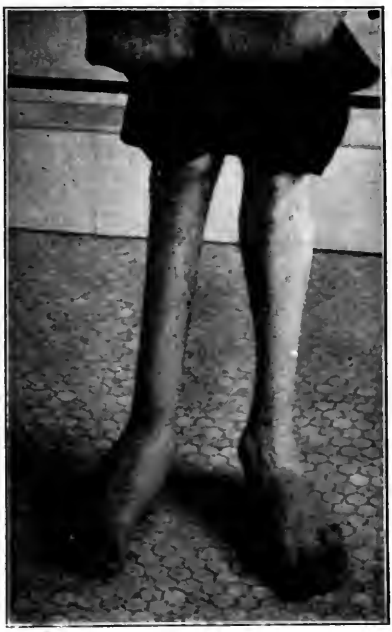


Fig. 127.—Postoperative club-foot. (N. Y. Hospital for Deformities and Joint Diseases.)

It is essential to ascertain the condition of the adjacent muscles. Movement should be studied in the uncorrected stage and the activity of the muscles recorded. If there are contractures the deformity should be corrected, and retention dressings worn for at least two months, when a second study is made of the force of muscular contraction. The muscle to be transplanted must be unaffected, and show no reaction of degeneration. Its condition can be

tested by a longitudinal incision through the muscle sheath; the healthy muscle is deep red in color, discolored if paralyzed, and a pale red if degeneration of the muscle fiber has supervened. This knowledge, a contribution from Hoffa, is very important in making the tendon transference successful.

Two methods of transference have been used: the first method employed was the suturing of the tendon of the healthy muscle into the tendon sheath of the paralytic muscle; the second method, however, has been found to secure a stronger and more permanent attachment for the new levator, whose cut end is inserted and attached to the periosteum at or near the insertion of the degenerated and useless mate.

Surgical Technique of Muscle and Tendon Transference.—The previously sterilized skin surfaces are painted with a 3 per cent. solution of tincture of iodine. After incision the lower part of the muscle and its tendon are freed from adhesions sufficiently to admit of free play, and it is made to assume a straight course to the new point of leverage. Shortening is attained by folding the tendon on itself. It is desirable to make use of the whole muscle; when this is impossible the tendon may be split not higher than the muscle belly, or interference in circulation and necrosis may result. One part of the split tendon is then attached to the new point of leverage, and the newly made muscle may in time functionate separately from that portion from which it has been ravished. Hoffa states that, as a rule, the two parts of the muscle continue to act together, and for this reason no part of the muscle should be so arranged that it antagonizes the main body. When linear freedom and proper tension are secured the tendon is reattached at the new site. Numerous methods are used for the periosteal insertion. Nutter bores a hole

in the bone from which the periosteum has been freed, and anchors the end of the tendon in this aperture. Wolff grooves the bone under the periosteum, sutures the tendon to it, and closes the periosteum over it. Sherman has made use of silver wire to suture the tendon to the bone. I have obtained uniformly good results by dividing the periosteum, suturing the tendon in place under it, and then driving a U-nail through the tendon and into the bone, at the same time using care to not strangulate the circulation in the tip. Dressings are applied in a position of overcorrection and a retention apparatus applied. To insure union and a good result it is necessary that an absolute asepsis be maintained throughout the operation, and the Esmarch bandage should be removed several minutes and free circulation in the limb assured before the dressings are applied.

It is understood that this operation will not correct an existing deformity. If the transplanted muscle is subjected to strain it will gradually relax. Preoperative correction, with the use of retention apparatus, should precede the transference by six or eight weeks. The aftertreatment is of equal importance. Overcorrection is to be maintained for a period of two months and support given for four succeeding months. Daily massage, or electrotherapy, and a gradual muscle re-education are to be initiated early in the third week. The retention apparatus must be constructed with this in view. A plaster cast can be cut in half for easy removal and reappliance.

The most satisfactory of the muscle-transference operations is the correction of a talipes equinus, secured by attaching the extensor proprius pollicis to the dorsal surface of the second phalanx of the big toe.

The results of tendon transplantation may be considered good. In paralytic affections of the leg and foot

great improvement can be attained, the motion may not be entirely free, and yet a marked advance over the former condition is secured.

4. THE APPLICATION OF ARTIFICIAL TENDONS AND LIGAMENTS.

It is now more than twenty years since Glucke first adapted some strands of silkworm gut as a bridge between the non-approximating ends of a tendon which had been severed by trauma. Five years later Kummel was able to demonstrate that silkworm-gut artificial tendons became converted into fibrous tissue. Lange, of Munich, made use of this valuable discovery and extended its usefulness; using strands of silk in place of the silkworm gut, Lange used the silk to reinforce atrophic tendons incapable of tension, and to extend healthy tendons which failed otherwise to approximate the new point of leverage. Thus, in paralysis of the quadriceps extensor with inability to extend the leg, Lange secured excellent function by utilizing the freed tendons of the biceps and semitendinosus, which were threaded with strong silk, which was in turn firmly anchored in the periosteum of the tubercle of the tibia.

Technique of Operation.—The field of operation, having previously been sterilized, is painted with a 3 per cent. solution of the tincture of iodine. The incision should be long enough to give a clear field. Protect exposed tendons and muscles from drying by covering with moist dressings or sterilized vaselin. Stitch the silk strands securely into the tendon; this is accomplished by a suture which extends at least two inches into the substance of the tendon, where a sharp U-turn is made and the suture is continued back to the point of entrance; the tendon is now provided with an extension of two long strands of silk. Long, strong metal forceps are used to pass or draw through the sub-

cutaneous fatty tissue the tendon and muscle which are to be transferred. The artificial tendon is attached to the new point of leverage on the bone by stitching it directly

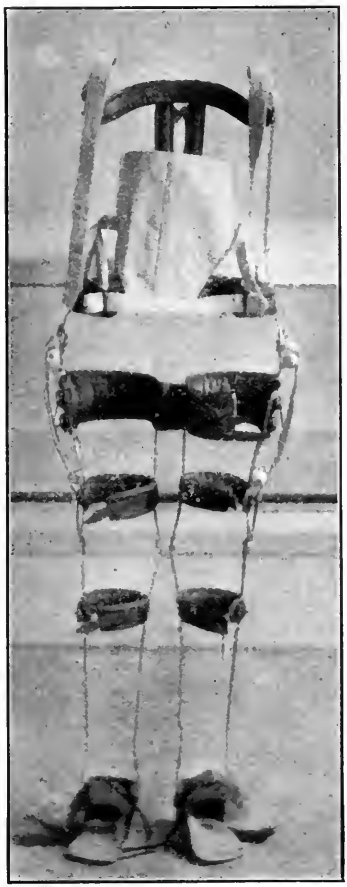


Fig. 128.—Frauenthal brace.

to the periosteum. In children it may be stitched through the cartilage, but never into the joint. The silk is prepared beforehand by boiling in a 1 : 1000 solution of bichloride of mercury for fifteen minutes, and subsequently in sterilized paraffin of a melting point of 155° for the same length of

time; before use the strands should be warmed to soften the paraffin. Number 14 braided silk is the preferred quality at our clinics. In the postoperative dressing the leg is adjusted to relieve tension as far as possible and plaster-of-Paris bandages applied over the sterilized dressings and cotton. This type of dressing is continued for about six weeks, and is followed by some mechanical protective appliance, which is retained for six or eight months and gradually dispensed with.

Lange, of Munich, has employed artificial tendons in 56 cases of tendon transplantation, of which 54 cases were successful. The silk tendons were found to be very durable, and after some months of use were found to be gradually acquiring a reinforcement of fibrous tissue, which steadily increased in thickness with the action of the transplanted muscles:—

> In the case of a girl in whom a silk tendon, made two and a half years before for paralysis of the quadriceps muscle, proved too long, an incision was made below the patella in order to shorten the tendon. It was easily found in the subcutaneous connective tissue, and was surrounded by a loose and movable connective tissue. There was no true tendon sheath. The tendon had the appearance of a bluish-white, tough, fibrous cord of the size of a large lead-pencil. Below, it was continuous with the periosteum over the tibia. The condensed surrounding tissue was two to three millimeters in thickness, and inclosed the silk cords, which appeared to be as sound as ever. Microscopic examination of an excised piece of condensed tissue around the sheath showed that in the deeper, and therefore older, layers next the silk the structure was identical with that of a normal tendon. In the superficial layers the structure was also tendinous, but with the addition of scattered vessels and fat-cells. (Lange.)

Bridges of artificial tendons usually heal promptly and give fair functional results. Artificial ligaments eight inches in length have been successfully employed. The strand of silk occasionally works out, and, as it has been

known to work out as late as ten years subsequent to the operation, it must be considered as a foreign body. Adhesions may grow, binding the silk to the adjacent structures, and thus causing limitation of motion; this is partly obviated by Lange's method of passing the artificial tendon through the subcutaneous fatty tissue. Vulpius considers that the systematic employment of artificial tendons is to be deprecated, and advocates a tendon-to-tendon anastomosis as the first condition essential to success.

5. ARTHRODESIS, OR JOINT STIFFENING.

The correct fixation of a flail joint, all of whose muscles have atrophied after paralysis, can be secured by mechanical apparatus, and also by artificial stiffening of the joint. Artificial ankylosis in a useful position, secured by operative interference, is known as arthrodesis. Arthrodesis is the method of choice of the members of the working class, who can ill afford the money and time required for the purchase and manipulation of expensive mechanisms.

The surgical stiffening of a joint has proved to be of most value for certain conditions of the ankle-joint, and must often be utilized for the flail knee; it has also been used for the paralyzed shoulder. It is not advisable to stiffen the hip-, elbow- and wrist- joint. Fixing a flail joint makes it stable, and thus provides for a return of part function in the extremity, and stiffening of a proximal joint provides a fixed point from which important unaffected muscles of the hands or feet take their leverage. Apparatus can thereafter be lightened in weight and finally dispensed with. The artificially splinted extremity is thereafter much more liable to fracture, and a stiffened knee, of course, prevents bending the leg when sitting. Indications for the selection of flail joints: (1) The very poor. (2) With failure of all other methods. (3) With no possibility

of regeneration of muscle. (4) When certain improvement outweighs certain disability. (5) Adult life, or termination of growth.

Time of Operation.—Vulpius, of Heidelberg, advocates an early operation for flail joint, considering that nine months after the attack the operator can determine that the case is hopeless without surgical interference. Tubby, of Liverpool, is much more conservative, and in his last volume (1912) advocates waiting for at least two years after the acute attack of poliomyelitis for possible restoration of paretic musculature. We are still more conservative in our advice in this particular, and believe that clinical experience justifies us in this course. In our clinic we have had numerous cases of infantile paralysis, of eighteen months and more, which have been passed on by other orthopedic surgeons as hopeless without the continued support of braces or operative fixation, in which we have succeeded in restoring some motor function. We have accomplished this by discarding all apparatus, whose pressure frequently interferes with nutrition of the levators of the joint, and prescribing a systematic course of physical therapy, including muscle re-education. It is our experience that neglect and the hasty assumption that an extremity is hopelessly paralyzed occur in direct proportion to the infancy of the patient and the indigence of the parents. Many cases, however, come from homes of prosperity with a history of the unavailing sacrifice of much time and money, in the hands of several attendants who have agreed that fixation is the remaining operation of election. The following case illustrates the foregoing:—

Male, Jewish American, 8 years of age. Onset September, 1909, with paralysis of both upper extremities, which receded from right arm, but left a left flail shoulder-joint. Treated by several physicians, who concurred in recommendation of artificial fixation, October, 1911, at infantile paralysis clinic of hospital, was found

to be a well-nourished boy with a totally relaxed left shoulder. Six months after treatment began, boy could raise left hand and arm extended in horizontal position; in one year the arm could be extended above the head and held for several seconds; the boy when last seen was steadily improving.

Age of Patient.—Unless absolutely demanded by the circumstances of the parents the mutilating operation of arthrodesis should be delayed until the patient has attained adult stature and growth has ceased. In particular the operation should not be undertaken before nor during the period of adolescence. This period is a great and well-recognized reconstructant of vitality of the whole economy, and no man can certainly state that a regeneration of atrophic muscles may not take place during the period. The orthopedist is further justified in putting a case over for a few years by the hope that the interim may engender a successful technique for nerve transference which will render a mutilating operation unnecessary.

We have the support of Lange, of Munich, who advises delaying this operation until adult life. Tubby, of Liverpool, and Bradford, of Mass., do not operate until 10 years of age; while Vulpius, of Heidelberg, would operate even earlier to secure ambulatory function, although he does not advocate arthrodesis of the upper extremity in childhood.

Technique of Operation of Arthrodesis.—Asepsis is to be maintained throughout this operation. The joint should be freely exposed to view. The cartilage of the articular surfaces is removed to bony tissue, with care to protect the epiphysial line. Careful approximation should be secured. Solid union is unnecessary if fixation is secured as soon as the skin sutures are in. Plaster dressings are left in place for two weeks, when the cutaneous sutures are removed, and thereafter for from three to six months.

6. NERVE TRANSFERENCE AND NERVE GRAFTING.

The clamor of a greatly augmented army of paralytics has recently stimulated research into methods for their relief. This is particularly true of the work being done in the great European clinics, where in the past ten years and more, particularly in the past five years, a constant investigation has gone forward in methods which may make practical the regeneration of paretic muscle, with a renewal of motor function. The paralysis of poliomyelitis is most often limited to a single muscle group or the muscles supplied by an individual nerve-trunk, while the contiguous nerve-trunk and the muscle groups innervated by it remain unaffected.

The regeneration and budding of a healthy peripheral nerve-trunk which had been severed accidentally or during an amputation has long been recognized, and many operators have endeavored to utilize this characteristic by grafting a portion of the healthy nerve-trunk into the non-functionating nerve-trunk of a partially paralyzed extremity. This operation, in one or two instances, has resulted in a speedy return of function to the paretic muscle; in many cases there was partial improvement after eight or more months, while in 50 per cent. of the cases there has been no return of function.

Theoretically, this operation of nerve anastomosis, offering as it does a possibility of regeneration to the spent nerve and the restoration of motor power to the paralyzed muscle, would be the operation of choice. While it cannot be recommended until an improved technique has greatly augmented the possibility of a successful result, the fact that one or two cases have given immediate and brilliant returns lends color to the hope that in a very few years it may be the operation of election.

A series of cases have been operated and reported by Tubby, of Liverpool; Spitzy, of Vienna, and Vulpius, of Heidelberg. Vulpius and his clinical assistant, Stoffel, have made an extended study of the anatomic basis for such nerve transplantations. They were able to demonstrate that the fibers which innervate particular muscle groups occupy a certain relative position and pursue a certain relative course in the nerve-trunk of conduction, and that this arrangement was almost constant in numerous preparations made by Dr. Stoffel.

Any operator who desires to make use of neuroplasty will wish to be familiar with the results of the Stoffel demonstrations. Dr. Vulpius's summary of the present stage of neuroplasty is as follows:—

"The operation (neuroplasty) is not as satisfactory in practice as it is attractive in theory. The results that have been obtained after much laborious work are certainly promising, but do not compare in point of efficiency with those of tendon operations. Not only does the latter insure the power of recovery in the muscles, but it is a very reliable proceeding, and one that justifies us in giving a fairly confident prognosis to the patient and his relatives. . . . . Where time and money are of little moment neuroplasty may reasonably be employed, but it is unsuitable for the bulk of hospital patients. . . . . Neuroplasty may be termed the *operatio divitum*, because it entails an expenditure of time and money, with frequently a smaller chance of success. The expenditure is, however, not unavailing, for the method represents a new line of advance, and it is our privilege and our duty to render it a practical, safe, and successful method. The task is a difficult one, but the problem is encouraging, and the successes that have already been obtained render our ultimate success certain." ("Treatment of Infantile Paralysis," Vulpius.)

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